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INTRACRANIAL HEMORRHAGE IN THE NEW-BORN

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OKLAHOMA CITY

The recognition of the intracranial hemorrhage in the new-born is exceedingly important. The diagnosis is fairly definite and the outcome is not always hopeless. Infant mortality in general has decreased, but this condition is still the cause of a large number of deaths during the first few days of life. Green found the condition present in 55 of 177 consecutive necropsies, or 31 per cent, performed at the Boston Lying In Hospital.

Etiology—Morse divides the causes into three groups—trauma, cerebral congestion and disease.

Formerly all cases of intracranial hemorrhage were thought to be due to trauma. This does not seem to be the case however, as the condition is found in cases with a strictly normal delivery. When the hemorrhage follows trauma, it is due to the overriding of the bones, causing a rupture of the sinuses, tearing the meninges or small veins. The trauma may be due to instrumental delivery, or to manipulation such as version and extraction. Abnormalities in size and shape of the pelvis may be the cause of the injury. Pressure of the mother's soft parts may cause the trouble. The importance of trauma is an established fact, as the death rate from intracranial hemorrhage is higher in the first born and in infants over the average size (Burpee).

In most cases there is cerebral congestion in the infant during delivery. The thin capillaries may give way, due to the increased pressure, and intracranial hemorrhage results. Asphyxia may play an important part here.

Disease—Syphilis has long been regarded as one of the foremost causes of intracranial hemorrhage. Except in the case of the premature, this is not now thought to be very often true. The hemorrhagic tendency in the new-born is a predisposing factor. Sharp and MacClaire are of the opinion that hemorrhagic disease is a less prominent factor than we formerly believed. Toxemia of unknown cause may play a part.

Symptoms—The symptoms, of course, depend upon the location and amount of the hemorrhage. The location may be any place within the infant's cranial cavity; supratentorial, infratentorial, or mixed. Rarely, it may occur in the ventricle or brain substance. Foot distinguishes two main clinical groups: (a) The rapid traumatic group, which is due largely to the rupture of a large vessel or vessels. Here, the symptoms come on soon after delivery and the bleeding tendency plays a minor role. The infant is cyanotic, respiration is labored, tremors develop in the arms and legs and death usually takes place within a few hours. If recovery does take place, spastic paralysis develops. (b) The slow spontaneous type due to an injury, which may be made dangerous through the tendency to bleed. This is probably the most frequent type. The symptoms do not appear until pressure is present. The child may seem to be normal for the first few hours and then it is noticed that he does not nurse so well. He becomes restless and cries a great deal. Often it is noticed that the tongue protrudes. Cyanosis is present and may be constant, but usually it is intermittent. Glasser states that when intermittent or paroxymal cyanosis is present, blood clots are invariably found around the medulla oblongata at autopsy. The intermittent cyanosis may, perhaps, be explained on the basis of pressure by the clots affecting the respiratory or vasomotor center or both. Congenital heart disease has to be differentiated, as it may be the cause of cyanosis. Evidence of cerebral irritation is noted, as there is spasticity, localized twitchings and perhaps convulsions. The fontanelle is usually bulging in hemispheric hemorrhage. The head is retracted in the infratentorial type. There may be a diminished coagulability of the blood. Carr in a series

of 200 new-born babies found the average bleeding time in normal cases to be two minutes and five seconds. The average clotting time in the normal cases was three minutes and two seconds.

Spinal fluid—The changes in the spinal fluid in cerebral hemorrhage depend upon the amount and type of hemorrhage. In a large hemorrhage the fluid is bloody. In basal capillary hemorrhage the fluid is xanthochromatic, but may be bloody and occasionally clear. The amount of protein may or may not be increased. Usually there are many red blood cells. A few red cells often result from the trauma of the lumbar puncture.

Diagnosis—Foot very nicely sums up the diagnosis—"In the first 24 hours after a rapid or forced delivery, or even after a normal delivery, irritability or extreme lethargy, disinclination to nurse, protrusion of the tongue; in the second 24 hours tenseness of the fontanelle, spastic twitchings of the limbs, intermittent cyanosis or paleness, all point to a progressive hemorrhage within the infant's skull."

Prognosis—Probably a large number of slight hemorrhages are not recognized and cause no symptoms. In a large hemorrhage, and especially in cases where no treatment is instituted, the mortality is high. In this group, if the patient survives, there is a spastic paralysis. These results may not show up for a few months. Later, mental retardation or epilepsy often develop. Some of the milder cases recover almost entirely.

Treatment—The most skillful obstetrical care will reduce these cases to a minimum. The general care of the baby is very important. He should not be moved from the crib for feedig, dressing or changing the diaper. He should not be put to the breast but milk should be expressed and given with a tube or Breck feeder. Care must be exercised that the body fluids are maintained. Lumbar puncture is done for two reasons—diagnosis and treatment. As a treatment, it relieves pressure and is repeated every eight to twelve hours as needed, according to symptoms. The fluid should be allowed to run off until the fontanelle is depressed, if distended, or until the fluid drops at the normal rate. The bleeding and coagulation time should always be determined. When the coagulation factors are deficient, blood should be given. Whole blood is given intramusculary 20 to 30 c. c. every eight hours for at least three injections. Citrated blood may be given intraperitoneally. The injection of the blood is given as long as needed and the treatment should be effective by the second day. Surgical decompression is not of value unless the site of the hemorrhage is localized. The general treatment and feeding should be continued with the child in the crib, disturbing it as little as possible for about ten days. At the end of that time, if the condition is satisfactory, it can then be put on the breast, and the usual care of the new-born instituted.

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THE UNDERWEIGHT CHILD*

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The problem of the underweight child confronts us most often between the years of one and ten. The greatest percentage is during the second year, then the third, fourth and fifth years respectively. The scale of growth during the first year is measured closely enough for practical purposes by the old rule: average birth-weight seven and one-half pounds, weight is doubled in five months, tripled in one year. The average breast-fed baby does this easily regardless of the unwise feeding procedures practiced all too often by mothers. In the artificially fed baby we have a far greater percentage of underweight children during the first year. Authorities agree that the chances of passing the first year are three times greater for the breast fed than the artificially fed baby in the poorer classes—where, of course, most babies are born these days. Artificially fed babies under the care of skilled pediatricians do as well as breast fed babies under the same care. The above takes into consideration all cases in the city and country where so many have no supervision other than the all-wise grandmother. We know also that Eagle brand milk and

*Read before the Southern Oklahoma Medical Association, at Chickasha, April 18.

similar preparations are large factors in the mortality of artificially fed babies during the first and second years. They contain two most excellent food constituents in large proportions—carbohydrates and fats, but are almost entirely deficient in the strength-giving and building factor—protein. They produce the fat water-logged, deceptively healthy looking babies to the untrained, who are almost one hundred per cent rachitic and whose resistance is so low that the least little infection produces a mortality.

In considering increases in weight we must bear in mind that the weight curve is greatly modified by heredity, race and body type. There are also seasonal variations wherein the greatest increase is from August to December, a less increase from December to March; and during the months from March to August there may be no increase in weight in a perfectly normal healthy child. The seasonal variations have been attributed to sunlight, diet, and the appearance of the respiratory infections during the latter part of the winter. Growth in length occurs from birth until the twenty-fifth year and often continues while the patient is sick so that the patient gets taller while he gets lighter. The normal weight for height is very similar for all races. A variation of minus twelve to plus twenty may be allowed for older children, and minus ten to plus fifteen in infants without any justification for alarm, provided foci of infection and other causes relating to metabolism are ruled out. Following is the scale of Wood and Gray, taken from figures of a large number of normal children:

Entering the second year, weight 21 pounds; height 76 cm. or 30 inches.

Entering the third year, weight 27 pounds; height 85 cm. or 33½ inches.

Entering the fourth year, weight 32 pounds; height increased five to seven inches per year.

Entering the fifth year, weight 36 pounds.

After the fourth year the weight increase is about four pounds until ten years of age. Seventy-five per cent of children are seven per cent or more underweight between the ages of two and ten, and sixty-two per cent of children are ten per cent or more underweight during these ages according to the figures of Emerson.

Rupe in examining 3000 children in selected cases found 10 in 100 with optimum weight. Less than 50 per cent are underweight in the upper and middle classes. The difference in nutrition and general conditions were shown to those of us who examined children recently in different districts in Ardmore. In one ward in which there were children predominantly from the poorer classes, I found evidence of rickets, postural defects and underweight in 85 per cent of children from five to seven years of age. In the ward opposite composed of children from much more prosperous families these findings were present in less than 50 per cent. Some of the most important factors in this underweight may be listed:

1. Poor feeding during the second year especially, and during the third and fourth years.

2. Various infections, chiefly respiratory.

3. Unhygienic environment — physical and mental.

4. Constipation and associated posture defects.

NUMBER ONE

So many families feel that after the first teeth are cut the child is ready to digest food on the table for adults. Eggs, gravies, meats—one to three times a day, greasy vegetables are piled into the little stomach to the exclusion of the great savior of childhood, milk, our most perfect food. All of us can remember the case of the six month-old child, presented in our senior year, who had thrived on adult food and was able to chin itself on the iron bar, at this tender age—just before it died of an infection easily overcome by a properly nourished child. From mild to pronounced forms of rickets can be demonstrated in these prize "Eagle Brand," "Malted Milk," "General Diet" infants in 95 per cent of the cases. This, as you know, shows in enlarged epiphyses, the well known rachitic beading, the rosary, or the Harrison's groove. Usually delayed dentition accompanies these conditions. They later succumb or are seriously invalided by the onset of the acute infections and respiratory diseases. If they survive the second year, the third and fourth are critical periods while the child's digestive tract strives to cope with the heavy and unsuitable diet. The struggle ensues until the natural time arrives for assimilation

of heavy foods. Then a few children begin to gain somewhat to make up for the valuable lost time. But too often it means a lifetime of poor digestion, chronic dyspeptics and delicate constitutions. Last year our colored maid warned us solemnly that we should likely have severe sickness in August in my own family — judging my 14-month-old boy by the experiences gleaned with poor feeding, universal among the colored, with its attending disastrous "second summer." This class is almost 100 per cent underweight. But all throughout the summer our boy gained steadily without loss of appetite, energy, or healthy vigor.

NUMBER TWO

Undoubtedly the most important factor for underweight is the onset of the acute infant diseases with masked and untreated complications and sequellae. Also the upper respiratory infections. Of these we may consider the ordinary acute colds or coryza. These are of two types,

(a) The contagious epidemic diseases, and,

(b) The non-epidemic colds due to lowered resistance of the mucous membrane.

Of the first and commonest variety the cause has been demonstrated as a non-filterable virus. This virus has been transplanted and found to produce typical "colds" without ordinary cocci at all involved; (b) the non-epidemic type is caused by the great increase in organisms due to lowered resistance from vessel constriction in m. membrane from chilling of any part of the body—especially feet. Immunity is not lasting from the virus types of colds. There is a great tendency to spread, causing otitis, virus infected bronchitis, and growth of adenoid tissue.

The incubation period is short, one to three days and treatment is unsatisfactory. Appetite is bad and the effects are lasting. Underweight children always result. In dispensary children, upper respiratory infections are present in 100 per cent of cases. And chronic tonsillitis is present in the vast majority of the classes so subject to upper respiratory infections. The most important causes of under-nutrition in children are chronic tonsillitis and chronic bronchitis. These are usually direct complications from acute colds. Lesser causes are nephritis, pyelitis, caries of the teeth and gum infections. The latter is rare; caries is a much abused focus of infection occurring very rarely in my experience. But pyelitis is not an uncommon cause of under-nutrition and is so often unrecognized, being treated by the parents and sometimes the busy doctor for malaria or worms. I have been treating several cases of pyelitis which have been most obstinate in clearing, by using routine alkalies, methenamine and hexylresorcinol alternately.

I have under treatment one child of excellent parentage, who is a post-scarlatinal sufferer. The specimens sent to the doctors were reported negative for one year. At this time a diagnosis was made by an ear, nose and throat specialist in a nearby city, who refused to do tonsillectomy until the pus was cleared up. The tonsils were enlarged and not healthy in appearance. After preparing her as well as possible in which her urinary cell count was reduced to four white blood cells to the hi-power field after two minutes centrificalization and clotting time reduced from ten to four minutes. Tonsillectomy was done. An examination six weeks later showed the first pus free specimen of urine. However, she has had three attacks of pyelitis since; two following otitis media (mild) and one following a simple rhinitis. She has been cystoscoped twice with ureteral dilation and no particular anomalies found. Colon bacilli were obtained by culture of pelvic contents from both kidneys. A vaccine was not made, in as much as little or no results have attended the results of B. coli vaccines in such cases. She is gradually overcoming her weight loss and I believe will ultimately be a healthy child. But only because she has wonderful home environments and parents who use every cooperative measure with their doctor and the specialists.

I have treated two children not very long since, who were both greatly underweight and both subject to "fever" and "bilious attacks," the "spells" lasting from two to seven days and taking all the energy for many days. Both had had tonsillectomies, both had large fragments of tonsils left on one side. Both had chronic parenchymatous nephritis, untreated for years. "Bilious attacks" is such a bilious expression and I rarely pass a day without hearing it said of some child whose liver is probably the only unaffected organ in its entire body. I wonder when we can learn that liver complaints are for the pro-

verbial "fat females and forty" and the alchoholics—not for tender buds under six or eight, or two years of age.

Another striking case I had recently under observation, was a boy from a neighboring town. He was 12 years of age, 14 per cent underweight and rather precocious in his development. He had come up to a local ear, nose and throat specialist for a tonsillectomy. In the routine examination before giving an anesthetic I discovered an enlargement of the heart and four plus albumen with many casts and pus cells in the urine. The tonsils were extremely bad. He was put to bed and given a modified Carrell diet for one week during which time the daily output of albumen decreased from 25 grams to five grams. There was no decrease of the casts and pus in the urine. Another week in bed with a slight increase in the protein and carbohydrate content of the diet gave a clear specimen except for a few hyaline casts. He was allowed to sit up for increasing periods during the day, and three and one-half weeks after the first visit he had a perfectly clear urine with a hemoglobin increased from 75 per cent to 85 per cent and the red count from 3,500,000 to 4,800,000. The tonsillectomy was done under gas and ether, the rapid Sluder technique being used. He made an uneventful recovery and was allowed to go home on the fourth day. Ten days later the boy was brought back to my office following bad effects attending the over-zealousness of the parents in having him up too much. There was a trace of albumin in the urine, the heart was not enlarged, but the pulse 112, hemoglobin had dropped to 70 per cent with the red cells again 3,500,000. This boy was sent home and will be confined to bed until the time that he shows a continuous normal blood and urinary and pulse finding. Having removed the focus of infection his outlook is bright for the future but no active exercise will be allowed for several months. In the immediate pre-adolescent stage too much attention can not be given the underweight child and a great majority of the cases the focus of infection can be found, removed and the greatest preparation for the adolescent stage can be obtained. This boy had been under the constant care of a busy practitioner, with parents extremely cooperative, but unfortunately had been treated only symtomatically. His gross pathology had been missed.

NUMBER THREE

Unhygienic environment plays an important part in the undernourished child. The physical side is accentuated in the poorer classes, giving the child a poor start in life. But especially in the cities and larger towns, and certainly here the "mental" side is important. Dorothy Canfield portrayed in a masterly fashion the change from a frail, weakly, nervous child to the opposite type in her book "Understood Betsy." The first care was given over to a wealthy spinster aunt, who devoured all books and literature pertaining to "How to raise your Baby," with most disastrous results to the victim child. Conditions were changed simply by force of circumstances, placing the child in the hands of a human, friendly, sensible woman, who did not regard Betsy as an absorbing experiment, but simply as another little human being who should think and act for herself. How we inwardly quake, and sympathize with the slender, nervous "scientifically raised" child, whose mother greets us with quotations from Holt and "the baby specialist at Chicago, or Dallas or what not." We have an unnatural condition to deal with demanding a sane, simple human treatment of the child as a child and not a laboratory for scientific study of human ailments. When I hear a mother tell me how delicate and nervous Virginia is (loudly before Virginia, who is proud to be so distinguished and nervous) I want to say with the doughboy back from France, when listening to his garrulous companions, "Aw, Hell!" But it is a factor, and chiefly in our best homes.

NUMBER FOUR

Rather as a combination of one or all of the above we have the last factor to be considered: Constipation and faulty posture. I found two correct postures out of all the children I examined in N. E. Ardmore, and only four in S. W., with really correct positions of standing. Practically all of these gave a history of constipation and poor appetites. I have under treatment now at least twelve children between the ages of two and six for constipation. The etiology is simple:

Of first importance is diet. Ingestion of foods insufficient in ash or not procuring proper bulk for the intestines are the chief factors. The bad habit of eating between meals also prevents proper rest for stomach or intestines with resultant intestinal disorders and constipation. The ice

cream cone, soft drinks and candy are factors of importance. Second: Mechanical causes, chief of which is faulty posture with the well-known lumbar lordosis, flat chest, winged scapulae and protuberant abdomen. This tends greatly to constipation with resultant under-nutrition. A third important factor is habit formation where the child is not taught to have stools at a regular time. This is an important factor, in the cities especially. The grand rush attending late sleeping hours, a hasty breakfast and away to school, leaves little time for proper bowel action. Lastly, drugs. And how often we find chronic drug addicts to the infamous Syrup of Pepsin and Syrup of Figs, or Castoria at the tender age of two, three and four years. They contain the most habit forming cathartics under a "natural" sounding name that achieves the purpose for which designated—namely, a heavy sale through out the country, and leaves an abnormal, constipated drug addict of three or four years.

Most of these postural defects can be overcome by energetic and systematic exercise in connection with the general upbuilding of the child's health. A few will require the services of an expert, and these few I always refer to orthopedic surgeons. One little child in particular, of six years of age, received a very marked benefit from a brace cleverly constructed to support hips, abdomen and shoulders. The entire construction weighing only a few ounces. This child took her exercise regularly and one year's attention altered her entire figure and standing posture, also completely cured her constipation.

Height-Weight-Age Table from Birth to School Age

Prepared by Robert M. Woodbury, Ph. D., Children's Bureau, U. S. Department of Labor

BOYS

Height (inches)	1 mo.	3 mos.	6 mos.	9 mos.	12 mos.	18 mos.	24 mos	30 mos.	36 mos.	48 mos.	60 mos.	72 mos.
20	8											
21	9	10										
22	10	11										
23	11	12	13									
24	12	13	14									
25	13	14	15	16								
26	----	15	17	17	18							
27	----	16	18	18	19							
28	----	----	19	19	20	20						
29	----	----	20	21	21	21						
30	----	----	22	22	22	22	22					
31	----	----	----	23	23	23	23	24				
32	----	----	----	24	24	24	25	25				
33	----	----	----	----	26	26	26	26	26			
34	----	----	----	----	----	27	27	27	27			
35	----	----	----	----	----	29	29	29	29	29		
36	----	----	----	----	----	----	30	31	31	31		
37	----	----	----	----	----	----	32	32	32	32	32	
38	----	----	----	----	----	----	----	33	33	33	34	
39	----	----	----	----	----	----	----	35	35	35	35	
40	----	----	----	----	----	----	----	----	36	36	36	36
41	----	----	----	----	----	----	----	----	----	38	38	38
42	----	----	----	----	----	----	----	----	----	39	39	39
43	----	----	----	----	----	----	----	----	----	41	41	41
44	----	----	----	----	----	----	----	----	----	----	43	43
45	----	----	----	----	----	----	----	----	----	----	45	45
46	----	----	----	----	----	----	----	----	----	----	----	48
47	----	----	----	----	----	----	----	----	----	----	----	50
48	----	----	----	----	----	----	----	----	----	----	----	52
49	----	----	----	----	----	----	----	----	----	----	----	55

GIRLS

Height (inches)	1 mo.	3 mos.	6 mos.	9 mos.	12 mos.	18 mos.	24 mos.	30 mos.	36 mos.	48 mos.	60 mos.	72 mos.
20	8											
21	9	10										
22	10	11										
23	11	12	13									
24	12	13	14	14								
25	13	14	15	15								
26	----	15	16	17	17							
27	----	16	17	18	18							
28	----	----	19	19	19	19						
29	----	----	19	20	20	20						
30	----	----	21	21	21	21	21					
31	----	----	----	22	22	23	23	23				
32	----	----	----	----	23	24	24	24	25			
33	----	----	----	----	----	25	25	25	26			
34	----	----	----	----	----	26	26	26	27			
35	----	----	----	----	----	29	29	29	29	29		
36	----	----	----	----	----	----	----	30	30	30	31	
37	----	----	----	----	----	----	----	31	31	31	32	
38	----	----	----	----	----	----	----	----	33	33	33	
39	----	----	----	----	----	----	----	----	34	34	34	34
40	----	----	----	----	----	----	----	----	----	35	36	36
41	----	----	----	----	----	----	----	----	----	----	37	37
42	----	----	----	----	----	----	----	----	----	----	39	39
43	----	----	----	----	----	----	----	----	----	----	40	41
44	----	----	----	----	----	----	----	----	----	----	----	42
45	----	----	----	----	----	----	----	----	----	----	----	45
46	----	----	----	----	----	----	----	----	----	----	----	47
47	----	----	----	----	----	----	----	----	----	----	----	50
48	----	----	----	----	----	----	----	----	----	----	----	52

SCIENTIFIC ACHIEVEMENTS OF THE ARMY MEDICAL CORPS*

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Military surgeons have, since early history, played an important role in the advancement of medical science.

Among these may be mentioned Dioscorides, a Roman Army surgeon during the reign of Nero; Saliceto, an Italian Army surgeon; Heinrich von Pholespeundt, of the Bavarian Army; Langenbeck, von Helmholtz, Krebs, Gaffky, von Esmarch, von Behring, of the German Army; and Pirogoff of the Russian Army. The names Pare, Laennec, Larry, and Laveran are familiar to all medical men; they contributed valuable work while members of the French Military forces.

The military surgeons of the armies of Great Britain and her possessions have contributed a long line of brilliant achieve-

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ments that have added to our store of knowledge; among these may be mentioned William Clowes, Peter Lowe, John Hunter, Sir John Pringle, James Lind, and, in more recent times, Leonard Rogers, Bentley, James, Christophers, Sirs Ronald Ross, Almroth Wright, David Bruce and William Leishman.

The medical corps of our own army can point with pride to its part in the advancement of medical science. Serving, as it does, in all sections of the United States and its possessions, during our various martial conflicts at home and in foreign countries, and with the field of preventive medicine constantly in mind, a stimulus has been present to encourage its members in the advancement of medicine.

"An introduction to the History of Medicine," by Lieut. Colonel Fielding Garrison of our corps, is one of the most thorough works on medical history by any English writer, and, from it, I have obtained most of the data presented herewith.

The records regarding our corps are not very complete prior to the Civil War. We know, however, that the first three Surgeons General of our army, Morgan, Shippen and Rush, all served in the Revolutionary War, and, at the time, were the most celebrated physicians in America. Benjamin Rush was an outstanding man, He was noted for his contributions to anthropology and insanity, his works being the first of their kind in America. John Hunter, a medical officer during the Revolution, contributed the first work upon surgery published in this country. General Hammond established the Army Medical Museum in Washington, that is now such a wonderful store of knowledge for the entire medical profession, and made valuable contributions on physiology and nervous diseases.

William Beaumont, (1785-1853) an officer of our corps, gave medical science its first definite knowledge regarding the physiology of digestion. While on duty at a frontier station in Michigan, a Canadian by the name of Alexis Saint Martin, was accidentally wounded in the abdomen. Beaumont cared for him, and after the wound healed a gastric fistula developed, through which Beaumont was able to study gastric digestion and the movements of the stomach. He accurately described the secretion of the gastric juice, the effect of the juice upon various foods, and the phenomena of inflammation of the stomach. His observations were published and the work became a classic.

Surgeons George A. Otis, Joseph J. Woodward, Charles Smart, and David L. Huntington prepared "The Medical and Surgical History of the War of the Rebellion" which was considered the greatest contribution to medicine and surgery of its time. Woodward was the pioneer of photo-micography in America and a leading pathologist; Otis, a surgeon whose operations upon the head of the femur and amputation at the hip joint were considered classical.

John Shaw Billings, (1838-1913) of our corps, created the Surgeon General's Library, which is now the second largest medical library in the world. He also originated the Index Medicus, covering the entire field of medicine. He was America's leading sanitarian and his work on ventilation is classical. After his retirement, he organized the New York City Public Library, certainly one of the greatest institutions of its kind in the world.

George M. Sternberg was the pioneer bacteriologist of America. He founded the Army Medical school in Washington and was Surgeon General of the Army from 1893 to 1902. His text book of bacteriology was standard throughout the country. He also wrote books on malaria and immunity. He discovered the pneumococcus and did the first work on the gonococcus in this country. It was he who exploded many of the fallacies as to the cause of yellow fever and made possible the work of the Yellow Fever Board, which he later appointed.

The work of Reed, Carroll, Lazear, and Agramonte, members of the Yellow Fever Board, is well known to every medical man throughout the universe. This Board proved the transmission of the disease by the mosquito, and an application of this knowledge by General, (then Major) Gorgas, and his associates, made the building of the Panama Canal possible, and has kept the Canal Zone free of the disease since 1905. General Gorgas later was Surgeon General of the Army.

General Farwood, Surgeon General directly after the Spanish-American War, was an authority on military surgery. He developed a special bullet probe and forceps that were used extensively.

To Colonel Russell, formerly of our corps, should be credited one of the out-

standing contributions to medical science. He demonstrated the protective value of typhoid vaccination, originated the technique of its preparation in this country, and was instrumental in making this a compulsory procedure in our army. His work has practically eliminated typhoid fever from our Army, a disease that had always played havoc in armies until the World War.

General Woodhull was the first to introduce the Anglo-Indian use of ipecac in the treatment of amoebic dysentery in this country. Colonel LaGarde was considered a national authority on gunshot wounds, and prepared a text book on the subject. Colonel Bushnell, a recognized authority on tuberculosis, contributed much work on this subject. Interest in helio-theraphy and medical ethnology was awakened by the studies of the effect of tropical light on blond races, by Lieutenant-Colonel Woodruff.

The late Lieutenant-Colonel Henry J. Nichols contributed much data upon "typhoid carrier" and prepared a text "Carriers of Infectious Diseases." His work with Lieutenant-Colonel Reasoner on the "Nervous Strain" of treponemae was very exhaustive.

To Colonel Ashford, formerly of our corps, we owe much of our present knowledge as to the cause of anaemia in the tropics. His work in Porto Rico on sprue and hook-worm infestation has done much to bring that country up to its present state of efficiency from a physical standpoint.

So far I have told of work accomplished by members of our corps that have served in the past. I will speak of some of the accomplishments of members who are still in active service.

Colonel W. L. Keller has contributed important knowledge in the field of surgery, especially of conditions within the chest. His vast experience with empyema following the World War, and his many stage operations in this condition has been an outstanding work. Major N. T. Kirk, in orthopedic surgery, especially amputations and bone grafts, has done much of benefit in changing former ideas and methods in this particular branch.

In the field of general and preventive medicine, we must think of outstanding men who are devoting their lives to the advancement of medical science. Colonels Craig, Siler, and Vedder, of our laboratory division, have contributed valuable work on malaria, amoebic dysentery, the demonstration of the filterable virus of dengue fever, its presence in the blood, and its transmission by a particular type of mosquito, beri-beri, etc.

The first work on Malta fever and the detection of its endemic presence in the Southwest was done by Ferenbaugh and Gentry of our corps.

The sterilization of water supplies by means of chlorine was very early applied by members of our corps and the present method in use for our troops in the field is by means of the Lyster Bag, devised by Colonel W. J. L. Lyster. Sanitary chemistry is a field assuming more and more importance from a military standpoint, and several of our members are at present devoting much attention along this line.

Aviation medicine is a recent branch of medical science, and many of our corps are now devoting their daily endeavors to this branch. We maintain a special school for teaching this subject, at Brooks Field, Texas, where new knowledge is constantly being added to our store.

New methods for the handling of the wounded and diseased in time of war are being originated at our Field Service School at Carlisle Barracks, Pennsylvania; and research problems in bacteriology, serum therapy, immunology, sanitary chemistry, etc., are being conducted in our Army Medical School in Washington, and by our Medical Department Research Board in Manila. Our pathological department is building up an exhibit at the Army Medical Museum in Washington, that is second to none in the world.

In the field of gas warfare, and the study of gas, both the toxic and non-toxic varieties, much is being accomplished in the laboratories at Edgewood Arsenal, Maryland.

Let us not forget to mention a contribution to medical science recently completed, "The Medical History of the World War," which represents the untiring efforts of several members of our corps over a period of several years, and without doubt, will receive the same praise as that occasioned by the Medical History of our Civil War.

New articles of equipment for medical, surgical, X-ray, and clinical laboratory work are being devised from time to time,

especially in the clinical laboratory field; and many standard articles in use in large laboratories throughout the country bear the notation "Army Medical School Model."

Time does not permit in this brief paper, the going into more detail regarding the scientific contributions by members of the Army Medical Corps, From the data presented, it suffices to say that we of the corps have a standard to maintain that has been set by our predecessors, and, with an *esprit de corps* that has been maintained throughout our entire existence, it is believed that many more valuable contributions will be made along the line of scientific achievements in medicine.

A COMPLEMENT FIXATION TEST FOR PELLAGRA*

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D. R. VENABLE, A.B., M.D.

Pellagra like syphilis is a most protean disease. Like syphilis also its origin is supposed to have been traced to the shores of America, from whence it is believed to have spread to the older civilizations of Europe, Asia and Africa and the islands of the seas. The history of this dread malady appears to be inseparably linked with that of maize or Indian corn. That pellagra is in some maner caused by the consumption of corn was the first theory advanced to account for the appearance of the scourge in Europe, and around this theory have centered the majority of the verbal, and sometimes acrimonious, conflicts that have been waged ever since by many students of the disease in many different lands.

Baruino, in 1600, described a disease existing among American Indians which may have been pellagra, and shortly afterwards Scipione described a similar malady. Both attributed the disease to corn, an important item in the diet of the American savage. About the same time a similar disease was observed among domestic animals that were fed spoilt corn.

It is a singular coincidence that the first scientific observations on pellagra in Europe were made by Gaspar Casal in 1755, shortly after maize imported from America had become a staple article of food in those provinces of Spain in which the malady was discovered. He named the disease Mal de la Rosa and in his published account of it he described "a peculiar kind of disease consisting of a combination of scurvy and leprosy". Antonio Pujati described the disease in 1755 after having visited Casal, and Adoardi gave to a similar disease which he observed in Northern Italy the name Alpine scurvy. The name "pelagra" was given this same malady by F. Frapolli of Milan and with a slight alteration in spelling, the term has been incorporated into the scientific literature of the world.

Subsequently, pellagra was recognized in many parts of Italy and in Roumania, Servia, Bulgaria, Austria, Hungary, Asia Minor and in Egypt. By 1856, the disease had increased in Lombardy to such an extent that 38,777 cases were found there. Commissions were appointed to study the scourge and even a pellagra asylum was established in which to treat victims of the malady. Dissensions arose as to the cause of pellagra, and while scientists and public officials quarreled over their theories as to its etiology, the disease increased with amazing rapidity until the high-water mark appears to have been reached in Italy between 1871 and 1884, during which time 104,067 cases of pellagra were reported in Italy alone.

In America, sporadic cases of pellagra were reported in New York and in Massachusetts as early as 1863, but the disease was not generally recognized until about 1907, when reports of cases began to appear independently from various Southern States. The disease had undoubtedly existed for many years in the South, but was not recognized as pellagra. In November, 1909, a conference on pellagra was held at Columbia, South Carolina, under the auspices of the South Carolina Board of Health. Since then the disease has been studied by various governmental commissions and by many independent investigators, both in this country and abroad. Champions of diverse theories as to the etiology of pellagra have appeared from time to time.

As early as 1839, Ballardino had demonstrated "beyond a peradventure" that the disease was due to the ingestion of damaged maize. He has been succeeded by many disciples and many dissenters. Pellagra has variously been held to be due to an aspergillus that grows on spoilt corn, to various bacilli from the same origin, to

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chemical poisons produced by fungi and bacilli, to the alleged fact that corn lacks proper nutritive value, to a diet lacking in certain vitamins, to intestinal parasites, especially the trichomonas hominis, to some parasite transmitted by simulium (the buffalo gnat), to drinking water containing colloidal silica, to some protozoon like the treponema pallidum of syphilis or the trypanosome of African sleeping sickness. But despite the many theories advanced and the painstaking research and laborious experiments of the best minds of the medical world and of scientists from related fields, the etiology of pellagra is yet to be discovered.

It is not within the scope of this paper to discuss in detail the many theories that have been advanced as the causative factors in pellagra. At the present time, most of the profession, in the United States at least, may be divided into two groups: those who believe that pellagra is due to a vitamin deficiency or some inadequate dietary, and those who believe that it is due to an infectious agent of some type. Those who oppose the vitamin deficiency theory do not accept the conclusions of Goldberger and his associates. Dr. Seale Harris in an address before the Southern Medical Association at New Orleans in 1920, offered several very pertinent objections to the methods adopted by Goldberger in his feeding experiments and these objections are concurred in by most of the physicians living in the communities in which these experiments were conducted. Briefly stated they are: first, the experiments were conducted upon long term convicts in communities in which pellagra was known to be endemic; second, the monstrous diet given these convicts over a long period of time was calculated to make even the most hardy ill and might be expected to produce scurvy in some cases; third, it is doubtful whether any of the six convicts who developed any symptoms even resembling pellagra really had the disease, as the findings were exceedingly meager.

In support of the theory that pellagra is due to an infectious agent are: first, its peculiar geographical distribution, being confined with rare exceptions to subtropical climates; second, the fact that in numerous instances epidemics of pellagra can be traced to one or two known pellagrins; third, the history of the spread of the disease is similar to the spread of other chronic infectious diseases; fourth, the remissions and exacerbations characteristic of most cases of pellagra are suggestive of parasitic disease; and fifth, arsenic has for many years been held to be almost a specific in pellagra as it is in other protozoon diseases such as syphilis and trypanosomiasis. To these we wish to add a sixth argument in favor of the parasitic theory, and this is that in untreated cases of pellagra the blood serum will give a complete fixation reaction.

It was after having repeatedly observed in blood cultures made on pellagrins spirillum-like bodies that we decided to attempt the preparation of an antigen with which to set up a complement fixation test for pellagra. The only mention in the literature of such a reaction being attempted in the diagnosis of pellagra that we have been able to find is the experiment of Lavinder. It seems, however, that his antigen was prepared from spoilt maize and not from blood cultures. Our blood cultures were made on a modified Hasting's egg media and the flasks were then exhausted of air and sealed. A grayish precipitate indicating a growth of some character could be noticed in the flask after some days. Spirillum-like bodies could be seen in this material with the dark field microscope, but these are believed to be due to the disintegration of the erythrocytes. In two different blood cultures taken on one patient, however, a spirillum was isolated and these cultures were kept alive for many months. One of these cultures was submitted to Dr. Hideyo Noguchi who kindly consented to give us his opinion on the type of organism we had isolated. He said in part, "The morphology of the organism does not suggest that of a spirochete, and I think there is no doubt that it is a spirillum". After the growth seemed to be fairly abundant, the culture was desiccated and part of it was extracted with ethyl alcohol and part of it with methyl alcohol. This alcohol soluble antigen was then titrated just as in the preparation of other specific antigens.

The complement fixation test is set up, following the Kolmer technic for the Wassermann reaction, the only difference being that pellagra antigen is used.

The Wassermann test is always run parallel with the blood to be tested for pellagra as, just in the complement fixation test for tuberculosis, a serum giving a strongly positive Wassermann reaction will also give a positive pellagra fixation test; one or two exceptions to this have

been noted, but they are so rare as to be negligible.

The blood serum of healthy individuals in whom no evidence of pellagra could be found gave perfectly negative reactions. A number of our colleagues were very considerate in furnishing blood from pellagrins for testing with the complement fixation reaction and it was found that the results obtained checked quite closely with the clinical findings. Some negative reactions were obtained in patients who were very manifestly pellagrins clinically, but in every instance it was found that arsenic (usually in the form of sodium cacodylate) had been administered for some time before the blood was obtained for the complement fixation test.

In one instance a four plus positive test for pellagra was obtained in a patient who was apparently in good general physical condition, except for some suspicious nervous manifestations. The physician in charge of this case stated frankly that he thought we had gotten a false positive in this patient. Much to the surprise of everyone, however, in a few brief weeks the patient suddenly began to lose weight and developed a profuse diarrhea and soon bloomed out with a typical pellagrous eruption and died in a short time, a fulminating case of pellagra with the toxic psychosis often accompanying such acute cases.

In a series of over three hundred cases in which blood was sent in for the Wassermann test, a pellagra fixation test was also run. In two hundred and eleven of these cases both tests were negative; in seventy-nine cases both tests were positive; in seven cases the Wassermann test was negative and the pellagra test positive, and in five the Wasserman reaction was positive and that for pellagra negative. Of the seven cases in which the Wassermann reaction was negative but the pellagra fixation test was positive, we have been able to secure clinical data in only four. The patients in two of these cases had been treated over a long period for syphilis and positive Wassermann reactions had been obtained several years previously. In the other two cases the patients were eventually diagnosed as pellagrins, and the physicians in charge of these cases did not even know that pellagra fixation tests had been run on the blood submitted for the Wassermann reaction.

In another series of cases, sera from 181 patients who had some symptoms suggestive of pellagra were run with the following findings: sixty-two were negative to both the Wassermann and the pellagra fixation tests; one hundred and six gave positive pellagra tests and negative Wassermanns; while thirteen patients had positive Wassermann and pellagra fixation tests. We have obtained the case histories of most of these patients and find that in all cases in which positive pellagra and negative Wassermann tests were obtained the patients presented clinical evidence of pellagra (although in some instances not very marked), and most of them were undoubtedly pellagrins. .Negative reactions were obtained in some few cases that were clinicallv pellagra, but in every such instance the patient had taken arsenicals over a long period of time before the blood was submitted for the fixation test.

From a study of these two series of cases, totalling about five hundred, we consider that we are justified in the belief that in the pellagra complement fixation test we have a valuable laboratory aid in the diagnosis of pellagra, and that it is a specific serum reaction for pellagra which is nearly if not quite as valuable as the complement fixation reaction for syphilis. The following brief case reports are selected from the cases just tabulated and are typical examples of the entire series.

CASE REPORTS

Case No. 1.—H. P., aged 7 years, white, was seen by us first May 15, 1925. Four years previously, he began to have an eruption on the hands and face, which finally involved the feet also and finally the elbows and knees. Constipation and diarrhea had alternated for some time, he having had a long attack of diarrhea three weeks prior to his visit to the Laboratories. His skin was rather dry over the entire body, but over exposed parts it had a reddish, beefy appearance, that over the hands and feet being badly cracked and fissured. The mucous membranes of the mouth were pale except the tongue was red and ulcerated. The blood Wassermann and tuberculosis fixation tests were both negative. The pellagra fixation test was four plus positive. The blood count was as follows: Reds, 4,300,000; Hemoglobin 80 per cent; Whites, 17,200; the differential leucocyte count: Polys 77, Small Lymphos 18, and Large Lymphos 5. The stool contained large numbers of trichomonas intestinalis and some blood. The urine was negative except for a trace of albumin.

Case No. 2.—Mrs. B. B., aged 42 years, white, dated her illness from August, 1926, when she bagan to have gastric disturbances and grew very nervous. Her mouth became sore and she began to have diarrhea and soon a rough, scaly eruption appeared on her hands, elbows and feet. She had lost considerable weight. She was markedly emaciated and there was a scaly red brown eruption over the backs of her hands and wrists and over the elbows, and her ankles and feet. Her tongue was red and beefy and so were the mucous membranes of her mouth. There was a tremor of her head and also her hands. Her mentality was considerably below par, and she was very emotional, crying easily. Clinical diagnosis pellagra. The blood Wassermann was negative and the pellagra fixation test four plus positive.

Case No. 3—Miss L. C., aged 15 years, had for two years been observed to be "off mentally" at intervals. Her appetite was at first variable, but finally she stopped eating and had to be forcibly fed. She lost weight and developed diarrhea. Her mouth was sore and red and there was a brownish red, scaly eruption over her hands and feet. Clinical diagnosis pellagra. The blood Wassermann was negative and the pellagra fixation test negative. This patient had had sodium cacodylate for many months prior to the taking of the blood for the fixation test, which undoubtedly accounts for the negative reaction obtained in her case.

Case No. 4.—Mrs. S. W. T., aged 52 years, had had obscure nervous symptoms and indigestion for two years prior to entering a sanitarium in July, 1925. At this time she had the typical "glove hand" eruption of pellagra and also a scaly, pigmented eruption over the neck, the pellagrin's "rosary". Her mouth was red and sore and her tongue beefy and fiery red. She was quite emaciated and had occasional severe attacks of diarrhea. She complained of headaches and seemed to be suffering from a confused mental state. Her appetite was poor and she slept poorly, requiring opiates. The blood Wassermann was negative and the pellagra fixation test four plus positive.

Case No. 5.—Mrs. M. J., white, aged 30 years, entered a sanitarium in October, 1927, complaining of nervousness, insomnia, weakness, headaches and nausea. She was despondent and somewhat hysterical. She complained of pain in her epigastrium and vomited at times. Her tongue was a beefy red and she had diarrhea alternating with constipation. She appeared to have lost some weight. She had no skin eruption, but there was a very bad odor to both the sweat and urine. There was no vaginal discharge. Clinical diagnosis pellagra. The blood Wassermann was negative and the pellagra fixation test four plus positive.

Case No. 6.—Mrs. I. N. P., 59 years old, had been unusually robust and healthy all her life until about three months prior to being seen in December, 1927, when she became very nervous and soon developed a morbid and persistent diarrhea. This was followed by a scaly, brownish red eruption over the backs of the hands and wrists and on the vulva. She became very much depressed and showed mental deterioration. Her tongue was fiery red. She finally passed into a comatose state in which she died. The blood Wassermann was negative and the pellagra fixation test four plus positive. The clinical diagnosis was acute pellagra.

Case No. 7.—W. L. C., 55 years of age, had lost 30 pounds in weight in the past three years. He had no appetite and was very much depressed, worried a great deal and suffered from insomnia. His tongue and mouth felt raw at times and his feet burned. He had a good bit of trouble with "gas on his stomach" and belched considerably. His skin was dry and scaly but there was no distinct dermatitis. However his tongue was fiery red, and with the mental symptoms and gastric disturbances suggested pellagra. The pellagra complement fixation test was four plus positive and the Wassermann was negative. The case is being treated as one of pellagra and is responding well to treatment.

Case No. 8.—Miss B. N., 37 years old, complained of burning and tingling of both hands and feet and was very nervous. She had a profuse diarrhea and her tongue was quite red. Her hands and feet were somewhat pigmented but there was no dermatitis. The pellagra fixation test was four plus positive and the blood Wassermann was negative. The clinical diagnosis was pellagra and as such the case is being treated with fair results.

Case No. 9.—Miss O. G., had lived in poverty most of her life and fresh vegatables and meats seldom form a part of her diet. In August, 1927, she first noticed that her skin was growing rough and scaly, especially over the neck, face and hands. Her mouth became sore and she be-

gan having burning pains in the vagina. In January, 1928, her neck, face and hands became acutely inflamed and her general condition much worse. She did not have any diarrhea and appeared to be fairly well nourished. There was an inflamed, pigmented eruption of a purplish-red color over her cheeks, the bridge of her nose, her neck and hands. The skin over the forarms and legs was also pigmented but not inflamed. The pellagra fixation test was four plus positive and the Wassermann was negative. The clinical diagnosis was pellagra.

Case No. 10.—L. L. D., 30 years of age, was a laborer and had been in good health until five or six years ago, when he began having headaches and sleeping poorly. He lost his appetite and began losing weight. He became very nervous and developed a morbid fear, as of some impending danger. He has attacks of diarrhea and constipation alternating. He has become very weak and unable to work. The pellagra fixation test in this case was four plus positive and the blood Wassermann was negative| The patient is being treated as a pellagrin.

Case No. 11.—W. H. M., 50 years of age, had had "stomach trouble" for ten years. He would have diarrhea and constipation alternately, and was very nervous so that he could not work at his profession as real estate dealer. He had no skin eruption but his tongue was quite foul. The pellagra fixation test was four plus positive and the Wassermann negative. The clinical diagnosis in this case was pellagra.

Case No. 12.—Mrs. L.W.C., 31 years of age, is married and is the mother of one child. For the past two years she had menstruated irregularly and was nervous and mentally distressed. Her family think "her mind is affected". She is very emotional and cries easily. She is usually constipated. The patient is thin and somewhat emaciated. She is very nervous and apprehensive. Her tongue is rather red but there is no skin eruption. The pellagra fixation test was positive and the Wassermann negative. The clinical diagnosis was pellagra.

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THE MALARIAL TREATMENT OF GENERAL PARALYSIS*

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Some four hundred years ago, Benvenuto Cellini, the eminent Goldsmith of the Renaissance period, tells quite candidly in his autobiography of his relation with Roman prostitutes, and frankly describes how he developed symptoms of a disease that cannot be mistaken as syphilis. He later tells how he came down with chills and fever, and declares that when he recovered from the fever the symptoms of his earlier malady had disappeared. Thus, we have the first recorded history of the treatment of neuro-syphilis with malaria.

During all the intervening years the treatment of syphilis has been along specific lines, with mercury and the iodides holding the lead. With the introduction of the arsenical preparations in 1910, by Ehrlech, a great forward step was taken in the treatment of syphilis of the central nervous system, especially, in the more acute cases and in the interstitial form where the spirochetes may be reached through the blood stream and be destroyed by direct action of the various arsenical preparations.

In the parenchymatous variety—that is general paralysis and tabes—the spirochetes become so deeply imbedded in the nervous tissue, which has such strong natural barriers to protect them from harmful agents, that it also protects the spirochetes from drugs introduced in the blood stream or spinal canal. Even in these cases some good results have been recorded. The physical condition of the patients improve, longer remissions are recorded and the average longevity of the patient is noted; but our death rate was little affected.

The non-specific treatment of neurosyphilis was developed in the clinic of the noted Vienna psychiatrist, Wagner-Jauregg. In his study of general paralysis he noted improvement in those cases that were ill with some febriie disease, such as typhus fever, pneumonia, erysipelas or typhoid fever. This caused Wagner to begin his experiments with Koch's tuberculin, typhoid vaccine, staphylococcus vaccine and various foreign proteins that would cause a rise in temperature. He found the cases so treated showed more improvement and had longer remissions than cases treated by specific methods. Not satisfied with the results obtained in the above, in 1917, he inoculated his first patients with tertian malaria. Since then the malarial treatment of general paralysis has been used extensively in the clinics and hospitals of Europe and America, and most favorable reports have been made in several thousand cases.

The malarial treatment of general paralysis is strictly empirical. Several theories have been advanced as to the action of the inoculation. Some investigators claim the high temperatures inhibits the growth or reduces the resistance of the spirochetes to therapeutic methods; again, it is maintained that the paroxysmal character of the disease—that is, the shock from chills—may have some special virtue not found in a continuous temperature. Another theory, and the one I think most reasonable, is that the malaria in some unknown way produces an immunity by the forming of antibodies in the blood to such a degree that the spirochetes are destroyed or their growth greatly inhibited. Other investigators believe the inoculation from malaria causes a great impoverishment of the blood which is followed by an active regeneration process that develops an immunity.

In injecting the malaria it may be given either intravenously or subcutaneously, most investigators prefer the latter. About 5cc. of blood is taken directly from the vein of the donor and injected in the arm, breast or scapular region of the patient. The injection is made deep into the tissue and the needle is moved in various directions from the point of the puncture to assure the transmission of the largest number of malarial germs. As the blood is infected at all times the stage of temperature of the donor matters little but it might be well to take the blood a short while after the initial chill.

The average time of incubation is seven

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to fourteen days, although, some cases will go twenty-one days, and frequently, cases will begin in five to six days. It is not uncommon for the inoculated patient to run a low grade temperature for several days before the initial chill. Although the tertian strain is used, the malaria does not always run the clinical course of the natural malaria. Most cases will have their chill every other day for two or three paroxysms when we see a change in the type, most frequently changing to the quotidian. In others the periodicity is quite irregular.

The patient should be allowed to have from twelve to fifteen chills with temperature ranging from 103 degrees to 106 degrees, or higher. Of course, the number of paroxysms that the patient is allowed to have will depend upon his physical endurance. Some patients will have only a few chills and be in a state of collapse and the malaria will have to be interrupted, but the majority will have the series without any difficulty.

We have few contra-indications for the use of malaria. Patients in the terminal stages of the disease should not be inoculated, also, patients suffering from heart or kidney complications. The lungs should be examined as a latent tuberculosis is likely to be lighted up by the malaria. Very obese patients should not be treated as sudden collapse is likely to occur. Very few complications occur during the course of the infection. A few cases of rupture of the spleen have occurred, jaundice, vomiting and diarrhea, herpes and paretic convulsions have been noted. In the latter the patients may die or it may be necessary to terminate the malaria, or, with small doses of quinine diminish its intensity.

A very interesting phenomenon of artificial inoculated malaria is the fact that it is exceedingly sensitive to quinine. We all know of the resistance of natural malaria to quinine and the large amount needed in many cases to eradicate it from the blood stream, but such is not the case in the artificial inoculation. Very small doses, in fact, in many cases, a five grain dose will terminate the chills and ten grains per day for three days will cause the fever to cease.

The question is often raised as to the danger of the transmission of malaria from the infected patient to others in the hospital. Several investigators have experimented on this line and the concensus of opinion is that after the blood has passed from three or four patients the gametocytes disappear from the blood and the malarial parasite no longer has an opportunity to complete the sexual phase of its life cycle in the mosquito and the parasite can only reproduce in the non-sexual form.

In experiments carried out to test the transmission of artificial malaria, mosquitoes were allowed to bite eleven patients infected with artificial malaria for 150 times, and after thirteen days to bite six patients free from malaria for 127 times. No malaria developed in any of the patients. However, when these same patients were infected with malarial blood they all developed a typical malaria. These same mosquitoes when examined microscopically, no sexual forms of the malarial parasite were found in their intestinal tracts and no sporocytes were found in the salivary glands of the mosquitoes. The conclusion was reached that it is perfectly safe to carry out the inoculation of patients with malaria even in localities where the anopheles mosquitoes abound.

The results from the treatment of general paralysis by malaria have been fairly uniform. In about 25% to 30% of the cases we can expect a complete remission and the patient will be able to return to work. Another 15% will show marked improvement and the ravage of the disease will be checked. In another 10% we will have some improvement and the patients will be able to care for themselves better and their conduct in the hospital will be changed from a disturbed, destructive and untidy patient to that of a quiet, obedient one. Five per cent of the patients will die either from inoculation of the malaria or from other diseases while undergoing treatment, leaving about 35 or 40% in which the treatment does not affect. These percentages apply to institutional cases and I believe if it is applied to earlier cases the percentage of arrests and improvements will be much higher.

It is interesting to note that of the nine patients first treated in the Vienna Clinic in 1917, four of these patients are alive today and carrying on their usual work. Two cases only showed slight improvement; another committed suicide while in a melancholy state a short while after the inoculation, one died in a paretic convulsion

during treatment and the ninth had a long delayed remission and many months after treatment was able to do manual labor.

Kirschbaum reported 196 cases in which he had 31% complete remissions, and 21% much improved, 11% slightly improved, 23% unimproved and 17% died. Kirby, of the New York State Psychiatric Institute, reports 141 cases treated with complete remissions in 32%, greatly improved in 14%, slightly improved in 10%, unimproved 25% and died 19%. Gesteman reports 294 cases with 38% showing complete remissions, 31% much improved and 31% not improved. Lewis, reporting 51 cases treated at Saint Elizabeth's Hospital, Washington, D. C., states 31% showed complete remissions. Green, of the Buffalo, N. Y., State Hospital, in a series of 93 cases, reports 26% with complete remission, 12% greatly improved and 22% slightly improved.

In the Eastern Oklahoma Hospital, we have inoculated 45 patients, of that number five are of recent date and will not be taken into the statistics. Of the other forty, eleven or 27% show complete remission, eight or 20% greatly improved, eleven or 27% slightly improved, ten or 25% not improved, which gives us a per cent of 74 that have been benefited by the malarial treatment.

In complete remissions we placed the patients that we feel are normal mentally and are able to return to their homes and sustain themselves at as high a social level as before the onset of the psychosis. The greatly improved, most of the mental symptoms have subsided and the patient is able to be cared for in his home and he is capable of labor but usually at a lower economical level than before the outbreak. In the slightly improved there is a diminution of the symptoms but the patients still require hospital supervision.

It is interesting to note the serology of cases of general paralysis treated with malaria. In about 50% we have some improvement, although, in some of the arrested cases we find no change either in the blood or spinal fluid. In the majority of cases the serological changes take place very slowly and the complete change may not take place for many months after the treatment is completed. The cell count is the first to react and in most instances there is a reduction immediately. The spinal fluid Wassermann is the most stubborn and in only about 25% of cases is it reduced to negative. The colloidal gold curve rarely becomes negative but is often reduced from a paretic to a luetic curve.

I believe all cases should be followed up with an intensive course of sulpharsphenamine or tryparsamide for many months. If so carried out we will note a still further improvement in the serology and a larger percent of negative spinal fluids. We will also note improvement in the mental condition of many of the cases that have shown some improvement from the malaria.

With an early diagnosis of spyhilis of the central nervous system and the inoculation of the patient with tertian malaria, to be followed by intensive treatment of the arsenical preparations, we may expect a higher percentage of complete remission. And may I suggest, that in all cases of primary syphilis a spinal fluid examination be made for it is now found that between 60 and 70% of all cases of untreated syphilis have an abnormal spinal fluid. Such cases should be treated until the fluid is clear. Only in this way can we prevent syphilis of the central nervous system.

NEURO-PSYCHIATRY AND INDUSTRIAL COMPENSATION*

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During a conversation with a judge who has had considerable to do with the Workman's Compensation Act, he said the prolonged disability so often seen following trivial injuries, was due in large part to the fact the workman was dissatisfied with his occupation. He was speaking particularly about coal miners and said many were compelled to remain in this employment by economic necessity and not from choice. Consequently they preferred two-thirds pay and idleness to full pay and an undesirable "job". I believe this quite a factor in prolonging disability, but not the only factor.

You, of course, are all familiar with the train of symptoms following trivial injuries that are classed as "nervous". They are all subjective as no objective signs can be found. I do not believe they should be designated by such a dignified title as "traumatic neurosis" as this leads the claimant to feel he is indeed a veritable invalid, it is

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such a high sounding name. Levy-Suhl regards as useless the various names—such as traumatic neurosis, hysteria, or wish-neurosis—using instead the blunt "indemnity neurosis". Director Weiler of the neurologic section of the Bavarian station dealing with neurotic invalids, feels that even the name "neurosis" ought to be discarded to avoid giving the laymen the impression that the condition of these persons is due to injury. He thinks "indemnity craving" would be a better term.

Just the other day a patient, not any way connected with industrial compensation said a physician had recently told him he (the patient) had a neurosis and he was under the impression this is practically an incurable condition. I explained neurosis meant only nervousness and he seemed much relieved. Let the patient become thoroughly and honestly convinced that he is an invalid and he is lost as a producer, and the compensation is lost, and you and I, as members of society, pay the bill. There is no reason, if the patient be properly managed, he should not, in many instances, be an economic asset instead of a liability.

These claimants usually complain of dizziness, weakness on exertion and say they have insomnia and sweat easily. Occasionally one has, he claims, a paralysis usually both sensory and motor, of an arm or leg or are filled with vague fears. Now, of course there are some who have had a severe brain injury with or without fracture of the skull, but history of the injury and symptoms are definite and leave no doubt concerning a real injury in the examiner's mind. Certainly if the injury produced no disability at the instant it happened, the chances are greatly in favor of a trivial result. The only exception is what is termed a late hemorrhage from trauma of some meningeal artery. Then the symptoms come on in twenty-four hours and are definitely irritative or paralytic, that is, they have local or general convulsions or paralysis. Then too, there are instances in which no injury to the head is claimed and yet the injured one says he has been totally disabled and cannot work because of weakness, dizziness and insomnia, with perhaps headache.

The theory that dissatisfaction with one's "job" is a prime factor in postponing admitted recovery, is borne out by information gained while interviewing several claimants. A glass worker spent a half hour detailing how the glass making industry had "gone to the dogs" since the advent of labor saving machinery and as he had some children who had about reached the end of adolescence and had some earning capacity, without actually saying so, gave me the impression that, unconsciously perhaps, he had decided it good time to retire as he was past middle age and saw prospects of etabishing permanent disability under the Workman's Compensation Act. The fact of the whole matter was, his injury was of so trifling a nature it could not have resulted in more than a few days' disability.

Another thing in which I do not believe, is routine spinal puncture in injured workmen. I have seen many claimants whom I am convinced had their alleged disability more firmly fixed in their minds because of this procedure and not a few who attributed new symptoms to lumbar puncture. It is a very valuable diagnostic and therapeutic measure, but should be used only in such instances in which the indications are plainly evident.

Now if a patient actually has a neurosis, it must be in one of three classes, hysteria, neurasthenia or anxiety neurosis. Simple "nervousness" will not do. The symptoms of hysteria, the so-called stigmata are too well known to physicians to require enumeration. Genuine neurasthenia is perhaps a little harder, at times, to recognize. It is an irritable weakness and has some somatic symptoms due to disturbance of the sympathetic system. Anxiety neurosis, the so-called psychasthenia, has its fears and phobias that come with startling suddenness and with more or less irregularity.

Naturally the first duty of a physician who is examining a patient who is said to have sustained an injury, is to find any organic change that is present and this requires a pains-taking physical examination with accompanying laboratory investigations. If there be any doubt, several sittings may be required to ascertain the presence or absence of any organic change. If none can be found, and the "nervousness" does not fall into one of the classes mentioned, it is proper for the examiner to report "no signs of disease or injury found". If any other disease is present, he should so report and state whether, in his opinion, the alleged injury was a contributory factor in its inception or exacerbation. I have stated above, dissatisfaction with one's occupation may be the cause of prolonging disability. There are others, a feeling of inadequacy, marital difficulties, conflicts with employers or fellow employees, and a host of others and I think

it a proper line of investigation the examiner should follow in determining the extent and cause of the alleged disability.

I have no reason to favor either the insurance company or the claimant, nor would I do so consciously, but certainly it is much better for all concerned to have the man return to work and thus save a good citizen to society instead of adding another non-producer. To me it is a calamity indeed, if a man is needlessly made an invalid and thus a liability instead of an asset. I have never had an insurance company to ask or in any way suggest I color my expressed opinion in the least. The physician should help the claim adjuster settle the case fairly, both to the claimant and the insurance carrier, but he certainly should get it adjusted as soon as possible and get the patient back to his regular occupation.

How can we aid in preventing such miscarriages of justice? First of all I believe the physician who gives first aid can do much by assuring the patient his lnjury is only of temporary nature if such be the case, and to tell him frankly he will be able to return to work in a short while. The claim adjuster, who often has lost faith in human nature, should retain his kindliness and sympathy for all unfortunates and not antagonize the claimant in any needless way. Then there should be a minimum number of physical examinations as the repeated enumerations of symptoms seems to impress them upon the mind of the claimant. Lastly, the physician, insurance company, and I may add, all the proper authorities should encourage the claimant to return to work as a therapeutic, sound, economic measure. I do not believe these patients should be placed in hospitals only under very exceptional circumstances.

THE CLINICAL SIGNIFICANCE OF PURPURA

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Purpura is of frequent occurence in a wide variety of clinical conditions, yet comparatively little is known of its true nature and significance. The relationship between blood platelets and purpuric manifestations was largely established by the work of Duke[1] some 20 years ago. We recognize now that many clinical forms of purpura are associated with platelet deficiency, but it is also apparent that this deficiency is not the only factor responsible for the purpura. Under certain circumstances purpura may be present with only a slight reduction in the platelet count, whereas under other circumstances the number of platelets may be reduced to 50,000 or lower before purpura appears. The course of idiopathic thrombocytopenic purpura before and after splenectomy emphasizes this point. The platelet count is usually low and purpura is a prominent symptom before operation.

Immediately following operation the platelet count rises and purpura no longer occurs. As time elapses the platelet count frequently drops well below normal, sometimes to a very low level, although purpura does not recur. The platelet count may be reduced experimentally by such agents as peptone[2] yet no purpura results. On the other hand, the number of platelets may be reduced by an antiplatelet serum and purpura can be produced in this manner[3]. The production of purpura by such a method is not an immediate reaction, but requires several days. Hyde[4] in 1928 demonstrated that if trauma to the tissue be added in addition to the antiplatelet serum, purpura would occur immediately. The trauma applied consisted in the mere shaving of the hair from the skin of the experimental animal.

It has been recognized that changes in the wall of the capillaries, commonly spoken of as capillary damage, might play a large part in the production of purpura. Such damage may be caused by a toxic substance circulating in the blood stream in which case the capillary change consists in an edema of the endothelial wall[5]. Purpura may be produced mechanically by such means as the application of a tourniquet which probably accomplishes this result by changes in capillary pressure.

Purpura or purpuric rashes may occur in many different infectious diseases. This association is most frequently seen with meningitis, measles, typhus fever and Rocky Mountain spotted fever. It rarely occurs with pneumonia, streptococcus and staphylococcus infection. Investigations by Julianelle and Reimann[6] have thrown interesting light on such processes. They have found that pneumococcus extract will produce purpura in laboratory animals. This property is common to various strains of pneumococcus but is not found in a number of other organisms such as the hemolytic streptococcus and green pro-

ducing streptococcus. The substance apparently has three distinct properties—it is hemolytic; it is thrombocytolytic and it will produce purpura. Heat will destroy thrombocytolytic property and its hemolytic property but does not destroy its ability to produce purpura. Mair[1] working along similar lines adds another possible factor in the production of purpura. He found that different mice showed a varying susceptibility to this purpuric producing substance. He was able to propagate strains of mice which were peculiarly sensitive to this substance. In other strains he was unable to produce purpura. Thus evidence is produced that a toxic substance may destroy platelets and produce purpura. These properties however are distinct and the purpura does not depend upon the reduction in the number of platelets. In addition there is evidence that some inherited susceptibility may play a part in the production of purpura. The occurence of purpura in certain infectious diseases is caused by thrombosis of the capillaries due to bacterial emboli.

There is a rare type of purpura which is probably due to some congenital or inherited defect. Hemophilia might also be considered in such condition. Whether the defect is in the blood or capillaries is not known. Many drugs and chemicals may produce purpura. Saponin destroys red blood cells and platelets and produces purpura[2]. Benzol, arsenic, iodine, quinine and atropin have all been reported as producing purpura. In some cases there is an obvious destruction of platelets associated with the purpura but as a rule no such change is demonstrated. Benzol will cause such a change in any individual whereas the other drugs mentioned produce the change in only certain apparently hypersusceptible individuals.

Purpura is frequently seen in non-infectious conditions which perhaps may best be termed metabolic disturbances. The most important of these conditions are rickets, nephritis, scurvy and arteriosclerosis. In these conditions nothing is known of the exact mechanism causing the purpura but it is agreed that local changes in the capillaries are propably the most important factors. The platelets are not reduced in this group of conditions and are thought to play no part in the production of the purpura.

Purpura, together with prolonged coagulation time and bleeding time, frequently occurs in cases of severe jaundice. The platelet count under such circumstances is usually within normal limits, although occasionally it may be somewhat reduced. It is quite likely that capillary changes play an important role in this type of purpura. On the other hand physical changes in the plasma due to the presence of an excess of bile salts may cause a tendency to purpura without there being any capillary damage. Bile salts will alter surface tension quite notably.

Within the past few years, interesting investigative work suggests that purpura may play a part in the response of the body to foreign protein invasion and allergy in general. In this entire group of clinical conditions the blood picture including the platelet count is essentially normal. This is the group of diseases to which Osler referred as the erythema group, and the clinical picture is characterized by purpura associated with visceral symptoms. The different clinical entities are known by a variety of names. Schonlein's disease is the name applied to that condition where purpura is found associated with joint symptoms. Urticaria or giant urticaria, is the term applied to that condition where purpura and hemorrhages from the mucous membranes are associated with urticarial wheals, Erythema nodosum is the name applied to the clinical condition where a deep subcutaneous nodule, frequently purpuric in nature, appear on the extremities associated with moderate fever and systemic reaction. Henoch's purpura is the term applied to the condition in which purpura is associated with abdominal pains. Alexander and Eyermann[3] have recently demonstrated the relationship between this condition and hypersensitiveness to certain food. That the different clinical pictures comprising this group may be very closely related, was demonstrated this past winter by a case observed in Barnes Hospital.

CASE REPORT

A young girl, age 19, was subjected to tonsillectomy because of repeated attacks of tonsillitis. Five days post-operative, she developed a septic temperature reaching peaks of 104 degrees. Her spleen became enlarged and she presented the picture of septicemia although no embolic phenomena were noted. Blood cultures were sterile. On the third day of the fever, which was the eighth day postoperative, she developed acutely inflamed joints. The joints involved were the left elbow and wrist and right knee. She presented at this time a typical picture of rheumatic fever except

for the enlarged spleen. The following morning she developed urticarial wheals which were confined to the swollen areas around the involved joints. The same evening she developed a purpuric lesion in the loose tissues beneath the left eye. She was given salicylates. The temperature subsided and the joint symptoms disappeared. The urticaria became widespread and was associated with angioneurotic edema of the upper lip, but disappeared entirely in two days. On the fourth day following the appearance of the joint symptoms, she developed acute abdominal pain and presented the picture of an acute abdomen so accurately that the surgeons were called in consultation. It was decided that she was probably a victim of Henoch's purpura and was further observed. The pain subsided within thirty-six hours and recovery thereafter was uneventful.

This one patient at different times presented the clinical pictures of rheumatic fever, Schonlein's disease, urticaria and Henoch's purpura.

Purpura occurring in the course of pulmonary tuberculosis offers an interesting field for speculation. We are coming to recognize many features of anaphylaxis in tuberculosis and it is just possible that purpura may at times be an evidence of such an association. Much has been written concerning the relationship between tuberculosis and purpura hemorrhagica. It is true that a clinical picture which cannot be distinguished from idiopathic thrombocytopenic purpura may occur in the presence of tuberculosis. Purpuric lesions, sometimes associated with joint symptoms but unassociated with platelet deficiency occasionally occur in tuberculosis. Purpura hemorrhagica has been produced experimentally by the injection of tuberculin in laboratory animals. There is some evidence that a simple non thrombocytopenic purpura may occur in tuberculosis which is similar to the purpura in the erythema group and probably likewise dependent upon anaphylactic phenomena.

In the foregoing part of this paper an attempt has been made to review briefly the factors which may be instrumental in producing the clinical picture of purpura. A summary may be made as follows:

Purpura may be dependent primarily upon platelet deficiency. Such a deficiency may be idopathic. On the other hand it may be due to some known toxic agent. This agent may be a bacterial toxin, a drug or chemical. The same toxin may produce purpura in one individual and produce no demonstrable effect in another, suggesting a difference in susceptibility of individuals. This difference is possibly an inherited characteristic.

Purpura may be dependent chiefly upon changes in the capillary wall or upon some abnormality in the blood aside from the platelets. Such a change may be an individual peculiarity and thus an inherited characteristic.

It may be due to a bacterial toxin, drug, chemical, or metabolic product. The change in the capillary wall may be related to some anaphylactic phenomena.

A combination of platelet deficiency and capillary damage may occur and the purpura result.

Purpura may be caused by embolic phenomena.

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THE PHYSICO-CHEMICAL INTERPRETATION OF CANCER*

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I feel that I should apologize for speaking to such a general audience on what may appear to be a technical subject. However, I predict that in the next ten years one will find it necessary to apologize if he attempts to discuss any fundamental biological subject without using a physico-chemical background. The human body is essentially a colloidal mass, and if anyone should know physical-colloidal chemistry in all its applications it is the doctor. The physiologists and those working with functional protoplasm have gone a long way out of their way in an attempt to avoid the application of colloidal chemistry. They have avoided the more simple consistent

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interpretation and have substituted an extensive nomenclature which only names conditions and gives no explanation for the observed facts.

In reviewing the very extensive literature appearing on the subject of cancer one is impressed by the fact that only an occasional paper appears touching on colloidal chemistry. Therefore it has been my object in the development of this paper to aid in establishing a consistent interpretation of the observed facts noted in the study of cancer. I wish to give a quotation from Dr. A. P. Mathews which expresses my attitude, "An examination of the colloidal state and particularly of the colloidal proteins is necessary for the understanding of vital processes".

The author has previously made several studies applying physico-chemical methods to the study of phenomena occuring in the circulating tissue, (blood). The present study was undertaken with the knowledge that facts observed in the circulating tissue may be applied to the fixed body tissue.

TISSUE GROWTH IN VITRO

When fixed tissue cells are placed in blood plasma and especially when such cells are crowded or the environment becomes stagnant, there is developed about the tissue an area of coagulation. This zone of coagulation is gradually removed and disappears about the areas of greatest growth. These observations have raised the following questions.

First, what are the forces active in producing the coagulation about the cultured tissue?; second, what is the mechanism by which the clot is removed?

What are the forces active in producing the coagulation? Coagulation of the plasma proteins is produced when substances change the colloidal proteins of the plasma producing a (1) Mass of low hydration capacity; (2) Maximum surface tension; (3) Maximum cohesion; (4) Isoelectric point.

It will be noted that tissue cells must be crowded together and the environment stagnant to properly promote the coagulation of the plasma about the tissue. When tissue cells are crowded with insufficient oxygen supply, there is developed a relative acid condition due to the accumulation of carbon dioxide. If the hydrogen ion concentration becomes sufficiently great, precipitation (coagulation) of the fibrinogen will occur in the form of hydrogen-fibrinate, the process being similar to the formation of hydrogen-casinate and hydrogen sterate.

The precipitation (coagulation) may also occur due to the contact of the two oppositely charged colloids. The tissue cells in a high carbon dioxide concentration (relatively high hydrogen ion concentration) will unite with the acid present to form electro-positive colloids, such colloids coming in contact with the electro negative colloids of the plasma will produce precipitation (coagulation) by establishing the four conditions enumerated above.

What is the mechanism by which the clot is removed? The clot is removed by the usual process of autolysis. The onset of such autolysis is delayed until a higher hydrogen ion concentration is developed. The fixed body cells do not undergo autolysis until the media becomes quite acid. The production of acid is increased due to the formation of the clot; the clot causes decreased oxidation and aids in the accumulation of carbon dioxide. As autolysis progresses there are created split products of protein, carbohydrate and fat hydrolysis. These substances act causing,

1. Increase in hydration capacity of the colloids.
2. Decreased surface tension.
3. Decreased cohesion.
4. Charges the colloid from the isoelectric point.

The tissue in contact fulfills these conditions and the increase in size accompanies the growth stimulus.

TISSUE GROWTH IN VIVO

The order of events in tissue growth in vitro was noted to be crowding, stagnation, coagulation and liquifaction with the liberation of growth stimulus. The same sequence of events occur in tissue growth in vivo. The following several lines of evidence point to the fact that cancer tissue develops in areas of suppressed oxidation, lowered metabolism and carbon dioxide accumulation.

1. Millet has reported fresh cancer tissue to be of pH 6.8,
2. In the pre cancer stage of tar cancer there is an increased acidity reported,
3. The surface tension of cancer tissue is less than normal tissue, and,
4. Radium emanations show a selective affinity of neoplasm.

All these lines of evidence point to the fact that growing cancer tissue differs in potential from normal tissue. This is further supported by the following observation: Tissue growing in vitro shows cell migration and changes in cell contour; these changes give the impression that motility is present. If the tissue cells are then subjected to X-ray emanation there is a complete cessation of all such motion. Some have considered that the cessation of motion is evidence of the specific action of X-ray. I am inclined to think the X-ray action is not specific but is only an illustration of an established isoelectric point. It will be observed that such discharged tissue undergoes great shrinkage with retraction. It has previously been stated that the isoelectric point yields: first, electrical neutrality; second, maximum surface tension; third, maximum cohesion, and fourth, lowered hydration capacity.

The growth of tissue in vitro is greatly increased by the presence of embryonic juice, the increase being so great has caused some authors to maintain that growth of fibroblasts is not due to the serum but to the small amount of embryonic juice within the tissue itself. It may be significant in this connection that foetal blood at half term has been shown to have a pH 5.8 at term 6. and six days after birth 7.; embryonic guinea pig skin growing best at pH 5.8 to 6.

It is generally understood that cancer has a special selection for age and location, also that irritation of certain organs predispose to cancer formation. Cancer occurs in the breast and uterus (two frequent sites of cancer development) at a time when there is involution, lowered metabolism, stagnation and carbondioxide accumulation. The transition of ulcer to cancer is common in the stomach and bladder, these organs being bathed in fluids which are definitely acid.

In order to avoid confusion it is necessary to consider three zones in any discussion of cancer: (1) Cancer tissue; (2) Zone between cancer tissue and normal tissue, and, (3) Normal tissue.

Any treatment of cancer must be developed with a knowledge of the presence of these three zones.

From the above discussion, one is led to believe that the principal method of combating cancer consists of prevention by: first, maintaining a high rate of metabolism in the tissue, that is a high rate of oxygen exchange; second, avoiding any condition which may produce stagnation and crowding of cells; third, the possible suppression of the zone of autolysis by the administration of a substance which liberates free oxygen in the tissue (such a substance is being tried at present); fourth, complete precipitation with asphyxiation of cancerous tissue leading to autolysis of the cancer. This last is being attempted by the administration of a colloidal coagulant with proper electrical charge. Also radiated auto serum may prove effective in sufficiently large doses as it has been demonstrated to concentrate in the cancer tissue.

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TUBERCULOSIS OF THE THYROID GLAND*

S. W. Budd, M.D. and Carrington Williams, M.D.

The authors point out the fact that pathologists, prior to the mid nineteenth century, thought the thyroid immune to the tubercle bacillus. As late as 1861, Rokitansky stated that tuberculosis never occurred in this gland. In 1862, Lebert described one of the first authentic cases. In 1865, Virchow expressed the belief that there was some antagonistic action between thyroid tissue and the tubercle bacillus, but was able to report three cases. Since this time the number of reported cases has increased steadily but the evidence goes to show that they are not numerous.

The miliary type is the most common and the authors suggest that a thorough study of the thyroid in cases of miliary tuberculosis might prove it to be more commonly present than we have thought. The disease may appear in any form; fibrosing, caseating or calcifying. Fistula of the neck leading to cold abscess in the thyroid has been reported. Infiltration of surrounding tissues and fixation of the gland make result.

The purpose of this paper is to report three cases of the sclerosing type, and to discuss the characteristics of this type. Practically all reported cases have come from French writers and it is suggested that this type may be more common among the French.

The thyroid is converted in part or in whole into fibrous tissue. The tubercle

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bacilli have not been found in this type but it is thought that the sclerosis is due to toxines in the blood arising from some remote tuberculous focus. As a rule the gland is small and hard but it may be larger than normal.

The author makes the following interesting comment:

The three cases reported here have much in common from both the clinical and the pathologic points of view. The patients were all women of approximately the same age (average, 41 years); all complained of soreness and tenderness in the region of the thyroid of a few months' duration; all three had slightly elevated temperatures and accelerated pulse rates; two had lost weight (average, 20 pounds); all were nervous, and two had increased basal metabolic rates. No positive signs of other tuberculosis were found in any of them, but one had had bronchitis for several years, and another had the scar of a cervical abscess and a chronic anal fistula. Pathologically all had the fibrous type of tuberculosis, which is the most unusual manifestation of the disease. The wounds healed in from two to four weeks; all gained weight (average, 10 pounds), and the general symptoms were largely relieved.

The diagnosis of sclerosing tuberculosis of the thyroid is important. Our first patient was suspected of having a malignant condition until a frozen section clarified the diagnosis. The second patient seemed to have an acute pyogenic infection and operation was delayed. The third case was diagnosed properly prior to operation because we had seen the other two so shortly before.

Tuberculosis of the thyroid is usually incidental to tuberculosis in other parts of the body; it is found more often at autopsy than at operation, but it is sometimes found when not suspected in adenomatous goiters.

The fibrous type, to which all of our cases belong, is easily confused. It has the same appearance as gumma of the thyroid but can be distinguished from it by the presence of pain, tenderness and fever. The Wassermann reaction should give a positive differentiation, and in the early stages there is likely to be an increased metabolic rate in tuberculosis and a rate of syphilis. It can be differentiated from acute thyroiditis by the less acute onset, less intense pain and tenderness, lack of increase in the leukocyte count, and the more chronic course. It is more difficult to rule out malignant disease, but the fever, tachycardia, tenderness and increased metabolic rate would suggest the inflammatory nature of the gland. If confusion exists even at operation, a frozen section of removed tissue will correct it. This differentiation, is of great importance, because the operation for malignant disease should be more radical than is necessary for tuberculosis.

The disease is a destructive one, so that in cases of long standing, lack of thyroid tissue may result in myxedema. In the early stages a mild degree of hyperthyroidism is likely to be present.

The treatment consists in the surgical removal of the diseased tissue as completely as possible, although, if a small amount of infected gland is left, the wound will heal and the body will take care of the remnant. This should be accompanied by the proper hygenic and dietary regimen for a tuberculous patient.

The prognosis for a local cure of the disease is excellent unless there is extensive invasion of surrounding structures. The presence of other foci of the disease determines the general outcome of the patient."

MECHANISM OF GALLBLADDER AND ITS RELATION TO CHOLELITHIASIS

Lester R. Whitaker, Boston (Journal A. M. A., May 14, 1927), summarizes his review of gallbladder function and activity as follows: The contents of the normal gallbladder are expelled by the activity of its masculature during the digestion and absorption of fat. Proof does not exist that this effect is brought about by a reciprocal action between the sphincter of the common bile duct and the gallbladder; in fact, there is considerable evidence against it. Stones can be produced experimentally in animals by interference with the normal mechanism for filling and emptying the gallbladder, resulting in stasis, and overconcentration of bile. It is possible that stones are produced in human beings by dietetic habits resulting in stasis. It is also possible that debilitating diseases, by reducing the muscle tonus of the gallbladder, favor stasis and the formation of stones. Stones can be forced out of the gallbladder in experimental animals by the giving of fat. It is probable that the same effect can be produced in human beings by the same means, provided the stones are not too large or the disease has not progressed far enough to render the musculature of the gallbladder ineffective.

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EDITORIAL

DANGERS OF THE SPINAL ANESTHESIA CASE

No type of anesthesia has so suddenly had such enthusiastic and justifiable reception as the spinal anesthesia. It will probably grow in popularity and deservedly so. Results of its use in proper and selected cases is brilliant, spectacular, satisfactory and safe, but a fatality resulting from its use is a tragedy, and often one which might have been averted. The successful use of spinal anesthesia depends first on the exercise of great judgment by the surgeon and anesthetist, if they are two individuals. The higher the field of operation, the more dangerous the procedure becomes; by common consent most men, that is good operators, stop at the level of the diaphram. Gall bladder, kidney and stomach surgery, while as a rule are nicely performed under this type of anesthesia, offer more obstacles and present more dangers than when the drug is so placed in the spinal canal that it only anesthetizes operative regions lower down. It would seem that for operating on the extremities, the lower abdomen and genitourinary system, there could hardly be any possible danger, but there is, the danger lies, not in the place where the anesthetic is being placed, but at the high point to which it might gravitate.

Administration of spinal anesthesia calls for intelligence, team work, and alert appreciation of danger signals. Constant blood-pressure readings are not only essential but they are the only means by which the anesthetist may be early appraised of the beginning of a dangerous situation. If the lighter than spinal fluid solution, that is the Pitkin, is used, the Trendelenberg position is the order of the day and it must be maintained. Even the drug should be administered with the patient in the prone position, on the side, and in a slight Trendelenberg, thus preventing its reaching the medulla.

Pitkin estimates that with the patient sitting, the spinal anesthetic can reach the medulla in eleven seconds, therefore with this solution nothing but the prone position should be used. The use of spinocain calls for the preliminary use of ephedrine but ephedrine is not nearly so useful and reliable in combatting blood-pressure fall as is epinephrine intravenously. Labat, Evans, and Babcock, all admirable authorities on this subject, advise (a) selection of the patient; (b) team work; (c) and necessary resuscitative combative material ready at hand, which may be used and flowing into the patient's vein in a minute or less. All authorities are aware that dangers must be early observed and must be met with the greatest promptness. With these things in mind it should not be forgotten that the lack of preliminary precaution and preparatiion is inexcusable and may result in a tragedy.

It is believed that for certain types of cases spinal anesthesia, properly administered, is one of the most remarkable achievements of modern medicine, but its administration cannot be trusted to the negligent or inexperienced.

VACATION PERILS

The United States has the vacation habit. In the last few years it has enormously increased. Transportation has been made so easy by the owners of millions of automobiles that access to mountains, lakes, rivers and streams is easy. Most of these trips present more or less dangers. Even the far off Rocky and Sierra Nevada Mountains, while practically free from malarial bearing mosquitos and similar hot weather pests, are potentially dangerous in a small way, in the possibility that Rocky Mountain fever may be acquired. Once on a northern California fishing trip the author recalls a general warning from the possible dangers from rabid wolves and coyotes.

To Oklahomans the first danger of an outing in the hills and along the streams in this State and in Arkansas is malarial infection. Lately there has been an enormous increase in this infection, no doubt due to the unprecedented rain-fall followed by large accumulation of stagnant water, which only evaporation could care for. It is well known that when a locality once becomes infected, if the medium of growth, the malarial bearing mosquito is present, the increase in infection of human beings is prompt, unmistakable and most difficult of control. To those who go on such trips, week-end or longer, the danger in selecting a camp site should not be overlooked. If these are in a malarial district and the camper is not well protected, malarial infection is almost sure to be acquired, and the length of morbidity is after that, problematical. The best protection is thorough screening; after which therapeutic doses of quinine nightly for a time are almost surely preventative.

Of course there are other dangers aside from the most common malaria. Those who are not protected by anto-typhoid vaccination may acquire that from water or from food served them at the thousand or more indifferent eating places. Here, of course, the ideal protection is vaccination. The smaller infections due to bites and stings are sometimes very troublesome. The dangers from snake bite seems to have increased, not because of an increase in snakes, but because thousands of people are tramping around where snakes are to be found. Antivenin is a tried and successful remedy, but moderate caution may obviate any necessity for its use. The woodsman should remember that venemous snakes are often in pairs and if one is seen, the unseen one is more often the dangerous one. Perhaps the best prophylactic against these is to keep your hands and face out of unknown places and wear high boots or puttees.

MEDICAL DEFENSE

For the information of our members it is decided to call their attention to the terms upon which Medical Defense is extended and available. At the out set it should be stated that formerly the Association undertook to pay all legal expenses. Gradually it was noted that some of our members attempted to include every other conceivable form of expense. The average amount spent in defending these cases was about $400.00. In one case more than that amount was expended and the next year the successfully defended member lapsed his membership and has never renewed. One of the earliest cases ever defended cost the Association an expense bill of more than $400.00; he too lapsed his membership and remained so. This was so disheartening that it was decided to limit the maximum amount of expense in any one case to not more than $100.00.

One of the most irritating phases of it was the palpable lack of good faith shown in some cases. In one case a member paid his dues, say on the 5th of May, was sued on the same date and expected the Association to defend his case, though his membership fee was not received until well along in June. This member gravely stated that the first intimation that he was to be sued was received the day or day after he paid his dues. Under the present rule—and they are positively binding—the following are necessities:

1. The member sued must have been in good standing upon the date of the alleged malpractice and *must have remained so continuously* (the only exception to this is that there is a month of grace, January of each year, in which the dues must be received in the office of the State Secretary).

An example of how a member may get in trouble under the above rule is this: He performs some service, say in 1928, he neglected to pay his dues, disregarding both notices from his County Secretary and the State Secretary, finally paying them in March or April, 1929. He is sued for alleged malpractice in May, 1929. He is not entitled to any aid, for his member-

ship expired December 31, 1928. Had he paid his dues as late as January 31, 1929, he would have been entitled to defense.

2. Malpractice must be alleged. This money was not intended to be used in defending any other type of action. Notwithstanding this it is not uncommon to receive requests for aid in other than malpractice suits.

3. Strictly speaking the Association should have prompt notice of a suit, a copy of the petition filed against the doctor and the names of his attorneys as well as those bringing the suit. Of course if the suit is merely filed and never comes to an issue and the attorney's bill is a nominal fee of $25.00 or $50.00, which is sometimes the case, the member is not entitled to the full amount of $100.00 to which he might otherwise be entitled. In other words if his attorney fee is $100.00 or more and is expended in good faith in defending a malpractice suit, he is entitled to reimbursement to that amount.

Editorial Notes—Personal and General

DR. C. G. SPEARS, Altus, is reported seriously ill.

DR. J. B. CLARK, Coalgate, is in New York City taking postgraduate work.

DR. J. H. VEAZY, Madill, has been appointed surgeon for the Frisco railroad at that point.

DR. A. L. MOBLEY, Muskogee, took two weeks' military training at Ft. Sam Houston in July.

DR. A. H. HATHAWAY, Mountain View, spent two weeks in June in Rochester, attending the Mayo Clinic.

DR. S. E. MITCHELL, Muskogee, spent three weeks on an automobile trip through Arkansas and Tennessee in June.

DR. and MRS. HOWARD S. BROWNE, Ponca City, spent two weeks in Denver, Colorado, where Dr. Browne attended an eye, ear, nose and throat clinic.

DR. WM. C. MILLER, Guthrie, spent several weeks in Denver, Colorado, taking a postgraduate course in the Colorado Congress of Eye, Ear, Nose and Throat.

DR. H. H. CLOUDMAN, Oklahoma City, has been named as full time health officer of the Oklahoma City schools. Dr. Eva Wells was selected as assistant.

DR. A. J. WEEDN, Duncan, spent two weeks in Atlantic City attending the international convention of the Protestant Hospital Association, June 13 to 17, and the national convention of the American Hospital Association, June 17 to 21.

THE STEPHENS COUNTY MEDICAL Society met in Marlow June 25, with Drs. C. N. Talley and W. Z. McLain as hosts. Dr. J. A. Hatchett delivered an oration on "Toxemia of Pregnancy," and Dr. C. M. Pounders lectured on "Summer Diarrhea in Children."

THE SOUTHERN OKLAHOMA Medical Association met at Medicine Park, June 27. The following program was rendered: "The Social Aspect of Tuberculosis," Dr. E. E. Darnell, Clinton, Okla; "Toxic Goitre," Dr. H. W. Livermore, Chickasha, Okla.; "Skin Diseases Commonly Met by the General Practitioner" C. P. Bondurant, Oklahoma City; "Treatment of Infections of the Hand" (motion picture film) by Oklahoma State University Extension Department. A fish dinner was served at 6 P. M.

THE STATE BOARD OF MEDICAL EXAMINERS, terms expiring in 1933, have received appointment by Governor Holloway as follows: Dr. W. P. Fite, Muskogee, regular; Dr. H. C. Weber, Bartlesville, regular; Dr. Frank H. McGregor, Mangum, regular; Dr. J. M. Byrum, Shawnee, regular; Dr. L. E. Emanuel, Chickasha, physio-medic; Dr. W. T. Ray, Gould, eclectic; Dr. D. W. Miller, Blackwell, homopathic. Dr. D. W. Miller, Blackwell, was made president, and Dr. J. M. Byrum, Shawnee, secretary. The personnel of the Board remained as formerly with the exception of Dr. Frank McGregor, who was appointed to succeed Dr. Harper Wright, formerly of Grandview, now of Oklahoma City.

TRANSACTIONS WOMAN'S AUXILIARY OKLAHOMA STATE MEDICAL ASSOCIATION.

The Auxiliary to the Oklahoma Medical Society met in the Huckins Hotel, Oklahoma City, May 29, 1929, with president, Mrs. W. K. West, presiding.

Five counties were represented by visitors; Oklahoma and Pottawatomie, the two organized counties, by delegates. There were forty-five in attendance.

Report of Officers:

Mrs. B. A. Hays, treasurer, reported $32.16 in treasury. Mrs. C. M. Pounders report of the work of Oklahoma County Auxiliary was interesting and inspirational. She reported forty-four dresses made for Childrens' Hospital; handerchief shower for girls in Home of Redeeming Love and other philanthropic activities. Report of Pottowatomie County was given by Mrs. T. D. Rowland of Shawnee: Hygeia has been placed in the Public Library and

High Schools of Shawnee. Last winter the auxiliary adopted the family of a disabled soldier and provided a joyful Christmas for them. Each month, a committee of two is appointed to provide books, magazines and flowers for ward patients in the hospitals.

The president's address by Mrs. W. K. West was interesting and constructive. She especially recommended and requested that we prepare and preserve for permanent record the early history and traditions of the pioneer doctors of Oklahoma and that a historian be appointed for the purpose.

Mrs. Irvin Abel, national treasurer, of Louisville, Kentucky, was introduced and in her talk made a plea for further state organization. She, too, requested the data of early-day practitioners and their interesting experiences.

Mrs. J. M. Byrum, Shawnee, was introduced and installed as president for the coming year.

Mrs. A. L. Blesh, Oklahoma City, gave a report of the nominating committee and the following officers were elected:

Mrs. Lloyd M. Sackett of Oklahoma City, President-elect.

Mrs. T. D. Rowland, of Shawnee, Secretary.

Mrs. S. E. Frierson of Oklahoma City, Treasurer.

Mrs. W. C. Bradford, of Shawnee, Historian.

Mrs. John Q. Mraz, Mrs. Horace Reed and Mrs. W. J. Wallace were elected as delegates to the meeting of the Woman's Auxiliary to the American Medical Society in Portland, Oregon.

Our Medical Society and State Board of Health have never at anytime invited our Auxiliary representatives to their health conferences.

MRS. T. D. ROWLAND, Secretary.

MRS. J. M. BYRUM, President.

EYE, EAR, NOSE and THROAT

Edited by Jas. C. Braswell, M. D.
1109 Medical Arts Bldg., Tulsa

Modern Cataract Surgery, Mills, L.: J. Am. Assn., 1928, xci, 1979.

Mills discusses postoperative iritus and prolapse of the iris.

Postoperative iritis is of four types: (1) traumatic iritis, (2) endophthalmia phaco-anaphylactica, (3) endogenous iritis, and (4) exogenous iritis.

Traumatic iritis is caused by rough or excessive manipulation of the tissues, irritation from hard fragments of lens remaining in the eye, tissue inclusions in the wound due to poor operative technique, pressure and drag on the incarcerated iris tissue, and a drag on the intact iris by herniation of the vitreous into the anterior chamber.

Endophthalmia phaco-anaphylatica may be prevented by careful expression and irrigation of loose lens cortex and in some cases by irrigation of the anterior chamber with warm half-normal saline solution (Reese), which gives definition to the lens substance that otherwise is not visible. Mills states that in his experience irrigation has never been followed by iritis.

Endogenous iritis develops from one to several weeks after any form of cataract operation as the result of unrecognized focal or systemic disease such as dental abscesses and intestinal infections.

Exogenous iritis is due to infection of the tear sac and bacterial invasion by way of tissue incarcerated in the wound.

Prolapse of the iris may be primary or secondary. Primary prolapse is due to prolonged fixation of the globe and iris following the knife blade through the incision. Secondary prolapse is caused by trauma due to awkward operative manipulations, excessive pressure, too small an incision, the pressure of defective dressings, meddlesome and too early inspection of the wound, strains and assaults during convalescence, defective incisions, delay of healing, and omission of iridectomy.

Until recently, the treatment of the incision in cataract surgery has been out of line with the treatment of other presumably clean operative wounds, i.e., full suture of the wound to prevent infection and restore the normal relations. Failure to suture the operative wounds of the eyes has been the chief single cause of infection from without and the extrusion of the intra-ocular contents. These complications may be avoided by covering the wound with a narrow but complete flap of conjunctiva formed during the incision or before, closing the flap over the sclerocorneal wound, and fixing it with about five interrupted sutures placed with regard to the peculiarities of the wound.

The Diagnostic and Prognostic Significance of Retinal Haemorrhage. Lamb, F. W.: Ohio State M. J., 1928, xxiv, 949.

Retinal haemorrhages may occur in any of the layers of the retina. Their anatomical location is an important factor in the prognosis as to vision.

Except in cases of obstruction or injury, the primary cause of retinal haemorrhage is disease of the blood-vessel walls. Retinal haemorrhages occur most commonly in nephritis associated with neuroretinitis. When there is a well-developed retinitis, the prognosis as to life is poor.

In arteriosclerosis, retinal haemorrhage is common and indicates that the blood-vessel walls are considerably weakened and that apoplexy is impending.

In diabetes, retinal haemorrhages are usually round and punctate and occur near the maculs. The prognosis for life is better than in albuminuric retinitis.

In leukaemia, the haemorrhages usually occur

in the fiber layer and near the periphery and have a white spot in the center. The prognosis is poor for vision and life.

Haemorrhages seen in the retina in a case of anaemia point to the diagnosis of pernicious anaemia.

In thrombosis of the central retinal vein, haemorrhages are exceedingly numerous.

When the diagnosis of choked disk is uncertain, a haemorrhage at the margin of the disk eliminates the doubt.

Retinal haemorrhages occur in from 30 to 40 per cent of newborn infants. In such cases they usually become absorbed without loss of vision.

An aid in the diagnosis and study of retinal haemorrhages is the use of the red-free light in the ophthalmoscopic examination.

Ocular Complications of Diabetes., Gifford, S. R.: Med. Clin. N. Am., 1928, xii, 423.

The best known ocular complication of diabetes is cataract, but the author believes that as a large percentage of diabetic cataracts occur after the fortieth year of age, a time of life when ordinary senile cataracts also develop, the importance of cataract as a complication of diabetes is overestimated. He states that a cataract should be regarded as a diabetic cataract only when it conform to the type occasionally seen in young diabetics. This type is characterized by the appearance under the capsules of both lenses of fluid vacuoles which progress rapidly. A condition allied to diabetic cataract is the occurrence of remarkable changes in refraction during the course of diabetes. This is probably due to a change in the osomatic pressure of the blood. The high blood sugar allows fluid to penetrate the capsule, causing the lens to swell, with resulting myopia.

Changes in the intra-ocular tension occur in diabetes. With high blood sugar and acidosis, hypotony is the rule. Two types of retinal lesions are seen: (1) white patches, which are usually small and single or occur in small groups, and (2) haemorrhages. It is probable that the arteriosclerosis which accompanies diabetes is an important factor in the etiology of the retinitis. The prognosis for life and vision is much better in these cases than in cases of albuminuric retinitis.

Otological Observations in Trauma of the Head: A Clinical Study Based on Forty-Two Cases., Grove, W. E.: Arch. Otolaryngol., 1928, viii, 249.

Grove states that persons who have sustained an injury of the head should be examined as soon after the accident as possible and at regular intervals over a considerable period of time.

The severity of the injury does not bear any direct relation to the development or degree of cochlear and vestibular symptoms.

Most injuries to the head in civil life are caused by a broadly acting force which compresses the skull with or without causing fracture. This compressing force results in damage to the brain, the cerebrospinal fluid, and the vascular system of the blood as well as to the skull.

In the skull, the compressing force of the injury finds its greatest expression at the base because of the more or less unequal strength of the constituent parts of this portion. The middle fossa, being weaker than the anterior or posterior fossae, is most often affected. The pyramid, weakly attached in the middle fossa, takes the brunt of the injury to the base and is frequently damaged.

Fractures of the temporal bone are divided into transverse fractures, longitudinal fractures, and avulsion of the tip of the petrous temporal bone. The longitudinal fractures are the most numerous. The labyrinth is damaged by the concomitant concussion and the fracture usually involves the middle ear and external canal. Transverse fractures cross the pyramid at right angles and completely destroy both the vestibule and the cochlea. Avulsion of the tip of the petrous bone is relatively rare.

The damage to the brain is caused by compression of the brain beneath the point of impact and at a point directly opposite. The damage done by the cerebrospinal fluid is caused by the compression of the lateral ventricles which sets the fluid in motion to expend its force in a whirlpool action in the fourth ventricle. The damage to the blood vascular system consists in a state of traumatic paralysis of the vasoconstrictors with resulting stasis of the circulation in the brain tissue, particularly in the central vestibular area annd probably also in the labyrinth.

In the temporal bone the chief findings at autopsy are haemorrhages. The intralabyrinthine haemorrhages are always perilymphatic unless the capsule of the labyrinth is fractured, in which case they may be also endolymphatic. The region most frequently affected by these intralabyrinthine haemorrhages is the scala tympani in the vicinity of the round window. The nerves may be torn or damaged by pressure from haemorrhage before their entrance into the pyramid, within the porus acusticus internus, or in the narrow bone canals leading to the endorgan.

The pathological changes in persons dying years after an injury to the head are atrophy of the nerve fibers, atrophy of Corti's organ, which is most marked in the basal coil, and complete or partial filling of the inner ear spaces and canals with hyaline connective tissue and bone.

The results of experimentation on animals show that the effects of mild injuries are the same as, though less marked than, those found at autopsy in the temporal bones of human beings, namely, haemorrhages in the inner ear, most marked in the basal coil of the cochlea and in the region of the round window, always in the perilymph spaces and never in the endolymph spaces. Degenerative changes are seen also in the nuclear territory of the eighth nerve in the floor of the fourth ventricle, affecting mainly the small cells of the nervus triangularis, the neucleus of von Bechterew, the tuberculum acusticum, and the posterior corpora quadrigemina. These changes are probably due mostly to vasomotor disturbances in this section producing stasis with subsequent destruction of tissue.

Haemorrhage from one or both ears occurred in nine of the cases reviewed. The author regards this as almost indisputable evidence of a longitudinal fracture of the temporal bone. It does not mean, however, that great damage to the function of the ear will necessarily ensue.

A cardinal symptom of injury of the vestibu-

lar system is vertigo. The vertigo is vestibular in origin if it has a rotary quality, if it comes on in attacks accompanied by nystagmus, or if it is produced by bending movements of the head. Other types of vertigo after injuries to the head are apt to be neurotic, especially if they are constantly present after the first two weeks or are accompanied by severe nausea, vomiting, and great mental excitement. The author is always strongly suspicious of a neurosis if the patient complains of a constant headache and vertigo after the first week or two following the injury.

Spontaneous nystagmus is another cardinal symptom of vestibular injury. It is unilateral or bilateral, and if bilateral, is more marked on one side. It is always rotary-horizontal in character. The author attributes it to a decomposition between the two vestibular systems. Bilateral nystagmus to the two sides is often seen in normal persons, but is always of the purely horizontal type, always equal in intensity, not associated with vertigo, and never influenced by the head movements test. The author has been unable to formulate any rule for the direction of the vestibular nystagmus in his cases.

Disturbances in the pointing reaction were present in twenty-eight of forty-two cases and constitute a part of the spontaneous vestibular symptoms which occur after damage to the vestibular system. Not much reliance can be placed upon them in deciding which side is involved. This is true also of the falling and Romberg reaction.

The irritability of the labyrinth to caloric stimulation was studied in thirty cases, all of which presented spontaneous labyrinthine symptoms. Normal reactions were found in ten cases, hypo-irritability in eleven cases, and hyper-irritability in nine cases. The irritability of the two sides was equal in twelve cases and unequal in eighteen cases. In the author's opinion, a difference in the irritability of the two sides, in other words, a decompensation between the two labyrinths, is of far more importance in the production of the spontaneous labyrinthine symptoms than hyperirritability or hypo-irritability.

The Rinne reaction was positive in thirty-five cases, negative in one case, and not recorded in six cases. Bone conduction was shortened in twenty-four cases, normal in six and not recorded in twelve. Traumatic deafness caused by injury of the head was found in thirty-one of forty-two cases. Complete deafness in one ear was present in one case. In a large proportion of the cases the defect in the hearing was bilateral, and a large number of the cases showed the upper tone range more affected than the lower and middle ranges. Of the thirty-one patients with defective hearinng, twenty-eight had symptoms referable to the vestibular apparatus

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The Etiology of Glaucoma., Duke-Elder, W. S.: Brit. M. J., 1928, ii, 236.

The first matter to be settled in the problem of intra-ocular pressure is the nature of the processes controlling the formation of the aqueous. The aqueous is not a secretion nor, under normal circumstances, a transudate. It is a dialysate of the capillary blood formed by the same processes as the other tissue fluids. The process is modified, however, by the relative impermeability of the ocular capillaries. The fluid contents of the eye must be kept clear and practically free from colloidal micelles. This is accomplished by making the capillary walls relatively impermeable. A dialysate in equilibrium with its parent fluid must have a very precise and definite chemical composition, osmotic pressure, reaction, electrical potential, and relationship between its hydrostatic pressure and that of the parent fluid. The aqueous in all conditions is in complete thermodynamical equilibrium with the plasma—chemically, osmotically, electrically, and hydrostatically. Its formation is a physicochemical process.

The second fundamental determination to be made in the study of intra-ocular pressure is the nature of the circulation of the aqueous humor. Three factors entering into this are: (1) a continuous metabolic interchange between the aqueous humor and the blood through the capillary walls; (2) internal thermal circulation caused by convection currents in the anterior chamber and, most important, a through-and-through pressure circulation; and (3) the changes in the volume of the contents of the globe which occur in the vitreous.

The vitreous is a gel bathed in aqueous. The main determinant of its volume is the degree of hydration of its colloid particles.

If glaucoma is considered merely as a pressure symptom, the two main factors in its etiology are: (1) a derangement of the capillary circulation involving a capillary dilatation which produces a rise in capillary pressure or increased permeability of the capillary walls which allows an excess of colloids in the fluids of the eye, and (2) changes of a physicochemical nature in the vitreous. These two factors act either alone or together, and their efficiency of the drainage channels in the region of the angle of the iris.

Primary Jugular Bulb Thrombosis, Maybaum, J. L., and Goldman, I. B.: Laryngoscope, 1928, xxxviii, 569.

In primary jugular bulb thrombosis an infected thrombus is formed in the lateral dome of the jugular bulb. In the early stages, the lateral and sigmoid sinuses are not affected. The authors review the clinical aspects of the condition and report nine cases.

Primary jugular bulb thrombosis should be considered in the diagnosis of cases with a history of middle ear suppuration, persisting septic temperature, and septicaemia otherwise unexplained. It occurs most commonly in young children. The temperature ranges from 99 to 104 degrees F. The organism responsible, usually a haemolytic streptococcus, can be easily recovered from the blood stream. Of considerable value in the localizing of the affected sinus is a differential blood culture according to the method of Ottenberg. This technique calls for culture from both internal jugular veins. The number of colonies grown from the culture of the sound side exceeds the number of colonies grown from the blood obtained from the affected side because the obturating thrombus prevents the passage of organism into the systemic circulation.

Prompt surgical interference is imperative. The operation should be done with minimal trauma. Ligation proximal and distal to the thrombus should be done and the sinus curetted and drained.

In the cases reviewed, operation revealed a sclerosed mastoid and an intact sinus wall which was gray and lusterless and contained an obturating thrombus.

The Pathology of Otosclerosis., Mayer, O.: J. Laryngol. & Otol., 1928, xliii, 843.

The author states that areas of otosclerosis are to be regarded as hyperplasias. This view is based not only on the histological appearance of the foci, but also on their multiplicity and typical and symmetrical localization, the presence of minute islands of atypical tissue (constituting the points of origin in these areas), the simultaneous presence of maldevelopments in the inner ear and other parts of the auditory organ, the general hyperplasia of the temporal bone, the association of the condition with blue selerotics and osteopsathyrosis, Paget's disease, and neurofibroma of the eighth nerve, and the hereditary character of the otosclerosis.

Vasomotor Affections of the Internal Ear., Portmann, G.: J. Laryngol. & Otol., 1928, xliii, 860.

The author states that the angiospasmodic syndrome of the labyrinth includes: (1) tinnitus, (2) deafness, (3) vestibular hyperexcitability, and (4) sympathetic hypertonia.

In addition to this syndrome of arterial resistance or hypertonicity of the labyrinth, there is the syndrome of hypotonia or laxity with the classical signs of the sensorial suffering, but with vestibular hypoexcitability and sympathetic hypotonia.

These two syndromes may alternate with each other. Different reactions of the vegetative system under the influence of various causes may be noted, but as a rule there is a hypertonic or parasympathetic syndrome which makes it possible to classify the subject as a vagotonic or sympathicotonic. In clinics there are seen fairly often persons in whom the disequilibrium seems to be caused by a global hyperexcitability of the vegetative nervous system. This state has been described as "neurotonia (Guillaume), "total disequilibrium of the whole system" (Laignel-Lavastine), "vegetative dystonia" (Sicard), and "amphotonia" (Danielopolu).

However, this vegetative dystonia may occur in persons who are predominantly vagotonic, in others who are predominantly sympathicotonic, and in still others in whom hypertonia predominates over the sympathetic at the level of one organ of the body and the parasympathetic predominates at the level of another organ.

Vagosympathetic disturbances and labyrinthine vascular spasms are due to most diverse causes. The causes may be mechanical, endocranial, toxic, or phychic. The most important factors affecting this regulating apparatus are undoubtedly the action of the nervous system and the action of the endocrine glands.

The Prevention of Nasal Deformities Following the Submucous Operation., Carter, W. W.: Arch. Otolaryngol., 1928, viii, 555.

Submucous resection is the best method yet devised to correct a deflected septum with obstruction. Certain precautions are necessary to guard against deformities. The operation should not be performed before the eighteenth year of age unless the indications are urgent. As the upper edge of the septum is an mioprtant part of the nasal arch, it must not be dislodged. The free edge of the quadrangular cartilage is an important vertical support. The septum should be removed by means of punch forceps without traction on the dorsal segment. Deformities resulting from this operation are best corrected by a conjoined bone and cartilage graft taken from the patient's ribs. Correction may be made also with bone or cartilage alone.

The article contains several photographs of corrected external deformities.

Intramuscular Injections of Bismuth a Specific Treatment for Vincent's Angina., Rigby, O. C.: Tri-State Med. J., 1928, i, 47.

As infection with the spirochaete of syphilis responds to treatment with arsenicals and also to injections of bismuth, it occurred to the author that bismuth might be equally effective against the spirochaete of Vincent's angina.

He first made a local application of 10 per cent acid tartobismuthate of potassium. The result was good. In December, 1926, he first injected 0.02 gm. of potassium bismuth tartrate with butyn. The injection was followed by permanent relief of the symptoms. The membrane disappeared and the smears became negative after twenty-four hours.

Rigby reports sixteen other cases with good results. In all, the smears were positive before the treatment and the symptoms ceased and the smears became negative within from twenty-four to forty-eight hours after the injection. The injection was made into the gluteal muscle. No local treatment was given.

The author states that a number of other physicians have had equally good results from this treatment, no failures being reported. The throat lesions apparently respond more satisfactorily than the infection of the gums.

UROLOGY and SYPHILOLOGY

Edited by Rex Bolend, B.S., M.D
1010 Medical Arts Building, Oklahoma City

A TREATMENT FOR CHANCROID

Rosenwald's method as abstracted by Brewer follows, the one thing of course to be dreaded in this line of treatment is the bubo, Rosenwald reports little trouble with them in the early stage. If there is as little trouble later, the time when most of us see them and it will shorten the time as reported. This method will be a great help.

A comparative study of 221 cases of undoubted chancroids shows under Rosenwald's method, a saving of 50 per cent in the days per man necessary to effect a cure.

Rosenwald's method for treatment is specific, easy, well within the province of the general practitioner, and can be compounded by anyone.

Two preparations are used:

Rosenwald's Suspension

Calomel	1 oz.
Zinc Sulphate	2 oz.
Tinc. Opium, Camphorated	2 oz.
Lime Water	8 oz.

Measure drugs by volume, not by weight. Mix the calomel and lime water in a glass stoppered bottle. Shake thoroughly several times a day for two days. Add the other ingredients, shake, and the solution is ready for use. It will keep indefinitely.

Rosenwald's Ointment

Zinc Oxide	1 oz.
Starch	1 oz.
Boric Acid	1 oz.
Gum Champhor	1 oz.
Carbolated Vaseline, 3%	12 oz.

Measure drugs by weight and make an ointment.

Treatment is carried on in the following manner: Clean the chancroidal ulcer gently. Take a thin piece of cotton, of a size sufficient to cover the lesion and vicinity, hold it over the mouth of the bottle of suspension and shake. Be sure the cotton is well moistened and covered with a film of the insoluble portion of the mixture. Place this upon the ulcer and pull down the prepuce to hold it in place. Leave this on for one day and then remove. Substitute a thin piece of cotton covered with ointment. Change this daily.

After the application of the solution the diseased tissue shows a bluish green coagulum, which separates from the sound flesh. In a day or two the dead tissue comes out leaving a clean wound which heals rapidly. Due to early sterilization of the ulcer, bubos are not encountered in early infections.

This treatment is as effective in treatment of chancroids in women, especially where the lesion is within the vaginal tract.

CLIPPINGS FROM UROLOGIC AND CUTANEOUS REVIEW

Most varicoceles are best treated by being let alone.

A negative Wassermann does not always mean a cured syphilis.

Let tabetic bladders alone as long as you can. They are easily infected.

Try sodium iodide intravenously in beginning spur formation as a result of gonococcal infection.

Open lesions or scars of healed ones over the tibia are highly suspicious of an old syphilitic infection.

Bismuth has been found of much value in the syphilis of young children who do not tolerate the arsenicals well.

In giving salvarsan injections do not use needles with any rust on them. Such needles leave telltale tattoo marks behind.

In the treatment of urinary extravasation there are two cardinal rules to follow: Divert the urine and lay open widely all extravasated areas.

Syphilitic disease of the heart with aortic insufficiency may cause death during the course of an anesthesia. It may also cause sudden death at any time.

Never remove any tumor of the scrotum until iodide of potash has been given in large dosage. Now and then these tumors disappear under such treatment.

Patients complain because their doctors give them scant information about their ailments, but it is a curious fact that the most successful doctors tell their patients the least.

In the acute renal infections of pregnant women be sure to try lavage of the renal pelvis and leave in the catheter for continued drainage, before inducing premature labor. Many women go through these crises nicely with this plan of treatment.

Conservatism in renal surgery is well worth cultivating. Many a kidney which at first examination seems to demand radical surgery will make a satisfactory response to well directed, patient endeavor of a conservative character. In recent years the absolute indications for radical renal surgery have been materially reduced.

It is to be deplored that ulceration, induration and renal adenitis do not immediately suggest chancre to every physician. All patients presenting this type of lesion should have a dark-field examination at once. By this procedure a positive diagnosis can usually be made long before the blood becomes positive or secondaries make their appearance.

IS DIRECT TRANSMISSIONS OF SYPHILIS FROM FATHER TO FETUS PROVED?

Johan Amnkvist believes that paternal participation in the infection of the fetus with lues is not impossible although it has not been definitely proved. In the author's series of 87 mothers who bore syphilitic children, 28 knew nothing of any luetic infection. In 23 of these cases, signs of syphilis were found, but in five there was no evidence of luetic infection—either clinical or serological. One of the latter cases was particularly interesting. Fourteen years ago this woman bore a 2950 gram child which presented a marked papular syphilitic exanthem and positive Wassermann. Clinical and serological investigations of the mother showed no evidence of lues. During the next fourteen years this woman was investigated serologically 36 times, always with negative results. In June, 1925, ten and a half years after the birth of the syphilitic child, she borne another child, having married another man. This child was quite healthy and free from all signs of lues, as was the mother. The author sees in this case a very probable evidence of paternal participation in the infection of the first child.

EFFECTS OF ALCOHOL ON BLOOD WASSERMANN

It is frequently stated that the ingestion of several ounces of alcoholic liquor will cause a positive blood Wassermann to become negative if the examination is made within 24 hours. If we assume that the patient's statement regarding the alcohol was true, this case proves at least that there are exceptions to the rule.

PYURIA IN INFANTS

According to Boddin, pyuria during infancy is not so much a disease as it is a symptom. This symptom may be due to various disturbances in the urinary passage, such as suppurative nephritis, congenital deformities, renal tuberculosis and the formation of stones in the pelvis of the kidney, ureter or bladder. The author gives a detailed report of one case. An infant, aged 5 months, was brought to the hospital appearing pale and weighing only a little more than at birth. Edema of the feet, hands and face were present. Later it also appeared in the region of the kidneys and in the genitalia. A few days later the child died. The post mortem examination revealed hemorrhagic suppurative nephritis, enlargement of the ureter, petechial hemorrhages in the trigon of the bladder, anemia, fatty degeneration of the liver and dropsy. The case was remarkable because the examination of the urine did not reveal the hemorrhagic inflammatory character of the disease. For this reason the case had been diagnosed pyuria, in spite of the fact that the edema indicated nephritis.

ORTHOPAEDIC SURGERY

Edited by Earl D. McBride, M.D
717 North Robinson Street, Oklahoma City.

Giant-Celled Tumor of the Neck of the Femur. Operation With Probable Cure. W. G. Turner. Can. Med. Assn. J., XIX, 342, (Sept.) 1928.

The patient was a nurse who was able to do her work until March, 1924. At that time she complained of pain and weakness in the right hip joint. At this time, there was some tenderness of the joint. The X-ray showed some rarefaction of the neck of the femur; but there was full movement.

In August, 1924, a pathological fracture occurred while the patient was turning over in bed.

On December 26, 1924, the hip joint was opened. The neck of the femur was found to consist of a soft shell from the acetabular rim well down to the trochanter, the fracture being felt about the middle of it. A flap of this shell was reflected, and the contents were found to be of a jelly-like consistency. This material was spooned out and the lining found to be smooth, except at each end. The ilium was exposed and a number of bone slivers chiselled off and packed into the cavity.

In March, 1925, a swelling was found below the trochanter. This was exposed and cleaned out.

An X-ray taken June, 1927, shows strong bone and moderate coxa vara.

Pathological examination of each specimen showed typical giant-celled tumor.

In June, 1928, the patient walked without pain, with slight limp and full range of movement, and could do a full day's work.

TUBERCULOSIS

Edited By
L. J. Moorman, M.D. and Floyd Moorman, M.D.
912 Medical Arts Bldg., Oklahoma City

Conservative Operative Treatment of Tuberculosis Arthritis. P. P. Vreden. Orthopedia i Travmatologia, V-VI, 10, 1927.

The treatment of tuberculosis arthritis, either conservative or radical, is not always satisfactory. A new method is being introduced at present which seems to be more promising. The originator of this method was Calot, the principle of it is a transformation of the cold, torpid, ischaemic tuberculosis process into an acute sterile one. He used injections of naphthol camphor into the tuberculosis focus.

The next step in this direction was the surgical procedure proposed by Dupuis de Frenelle and Robertson Lavalle's method and proposes the following surgical procedure: Through a small incision of the skin a long narrow gauge is introduced and driven through one and the other articular extremities until it fixes the joint. A graft is then taken from the crista tibiae together with the periosteum; the length of it is equal to the part of the gauge driven into the joint. The gauge is removed and the graft is inserted in the previously made canal. The skin is sutured with one suture. A plaster cast is applied for from four to six weeks. In cases with muscular spasm, a stretching can be made while the patient is under anaesthesia. The presence of sinuses and even free pus in the joint does not constitute a contraindication.

The immediate result of the operation is an acute local reaction with complete disappearance of pain and reestablishment of motion in about two months after operation.

The Treatment of Tuberculosis by Heliotherapy. Horace I. Grasso. Radiology, XI, 217, (Sept.) 1928.

The beneficial results obtained in treating cases of surgical tuberculosis at the J. N. Adams Memorial Hospital and other institutions where heliotherapy is practiced, have far surpassed anything ever accomplished by the use of surgery.

Essentially, the treatment used is patterned by Rollier with some modifications. Exposure should be made in open air, with nothing to interrupt the sun's rays. South porches, screened against prevailing winds, are best. The whole body, with the exception of the head, should eventually be irradiated at one time for proper treatment. The initial treatment consists of five minutes' irradiation of the feet, repeated three times during the day. The irradiation is then extended, five minutes being added daily to the isolation period of each previously exposed area. A suitable maximum is three hours, an hour and a half, morning and afternoon.

The method of sun-cure dosage in terms of hours and minutes is open to severe criticism, so during the past two years a more rational system of dosage, based on sunlight intensity, has been adopted. As a rule, the patient's tolerance of sunlight can be estimated from the pigmentation of the skin. Contrary to general opinion the author believes that sunlight judicially applied in pulmonary as in surgical tuberculosis, particularly so in hydropneumothorax and pyopneumothorax

cases. He also condemns the use of artificial light substitution for heliotherapy, although he admits the value of lamps during the cloudy season. Without the tonic effect of moving air on the naked body, regular hours, suitable diet, and above all, rest, light rays are robbed of much of their beneficial action.

Extensive statistics covering the 1160 patients discharged, up to January 1, 1927, with extra-pulmonary tuberculosis are quoted. Of the 355 cases of tuberculosis of bones and joints: 198 or 55.8 per cent were discharged as apparently recovered; 85 or 23.9 per cent were discharged as arrested; 34 or 9 per cent were discharged as improved; 24 or 6.8 per cent were discharged as unimproved; and 14 or 4 per cent died.

No patient who had been confined to bed was discharged apparently recovered, until he had been up and about for at least six months, if the lesion was in a bone or joint.

Two Cases of Chronic Deforming Arthritis Due to Hereditary Syphilis. L. Ricciardi, Pediatria, XXXVI, 395, (Apr.), 1928.

The author reviews briefly the rich nomenclature of the articular alterations of infancy and recognizes the obscurity of the clinical picture of the chronic arthropathies due to the scarce knowledge of the etio-pathogenic factors of the disease. He then illustrates two cases of deforming rheumatism of luetic nature, in which the articular manifestations appeared late. He cites the belief of various authors regarding luetic arthropathies and illustrates the clinical picture, recalling the Fournier held that thirty-nine percent of hereditary luetics became subject to articular syphilis, while von Huppel, Stein, and others consider the percentage to be fifty-six percent. Hydroarthrosis manifests itself very frequently between the ages of eight and fifteen years. The author closes with the conclusion that chronic deforming rheumatism of infancy is often due to hereditary lues, but having onset, symptomatology, and evolution like other forms, it is necessary to establish at the earliest possible moment its nature, in order to intervene energetically with specific treatment, it thus being possible to avoid deformities.

Tuberculous Cervical Adenitis. Richard H. Miller and William M. Shedden. The American Review of Tuberculosis. May, 1929.

"Tuberculous lymph nodes of the neck constitute a focus of disease which, early in its existence, may set free tubercle baccilli that are carried about the body, resulting in a mild bacteriaemia. The bacilli may come to rest anywhere in the body, and may give rise to later secondary loci of disease. The bacilli may also extend from the originally infected nodes and invade contiguous ones, finally involving most of those on one or both sides of the neck. Anything which builds up and improves the patient's general condition, such as rest, hygiene and heliotherapy, increases his power to combat the original infection and also to overcome its possible secondary manifestations. Ultraviolet light is a valuable factor in building up local resistance, and it is particularly valuable in its good effect in the healing of sinuses; it does not seem to have much effect on lymph nodes of any considerable size. The Roentgen ray is, as Hanford has shown, very valuable and must be accepted as an important method of treatment. Tuberculin is of doubtful effectiveness, but seems to help in some cases.

"It seems to us that any form of treatment which is directed against the swollen lymph nodes, and fails to take into consideration the fact that the nodes contain living bacilli, which have been, may at the present time be, and probably will in the future be set free into the general circulation, is losing sight of the most important fact in the whole condition. In addition to the cases reported in this paper, we have seen and followed hundreds of cases of tuberculous cervical adenitis, and we are impressed with the number of those which present distant lesions in the body, to say nothing of the deaths, secondary to lymph nodes in the neck."

"We therefore wish to go on record as believing that in a considerable number of cases of early tuberculous cervical adenitis, whether in children or adults, and often in cases of longer duration in which operation is technically feasible, radical surgical operation is the method of choice. We do not fear the possibility of scars, because in the hands of a good surgeon they are not as bad as they used to be, they can be improved by a simple cosmetic operation, and we prefer a scar in the neck to later tuberculosis of the eye or the lungs. The operation must be supplemented with all the supportive and palliative measures which may be used."

BOOK REVIEWS

The Nose, Throat and Ear and Their Diseases: In original contributions by American and European Authors. Edited by Chevalier Jackson, M.D., Professor of Bronchoscopy and Esophagoscopy in the University of Pennsylvania, in the Jefferson Medical College, and in the Graduate School, University of Pennsylvania, and George M. Coates, M.D., Professor of Otology, Graduate School, University of Pennsylvania. Assisted by Chevalier L. Jackson, M.D., Assistant in Bronchoscopy and Esophagoscopy, University of Pennsylvania. Octtavo volume of 1177 pages with 657 illustrations and 27 inserts in colors. Philadelphia and London. W. B. Saunders Company, 1929. Cloth, $13.00 net.

This is a beautiful work. Chevalier Jackson and his associates are authorities on bronchoscopic work. This volume is unusal in that it is composed largely of contributions from the leading American authorities of affections of the nose, throat and ear. It is divided into six parts, each part of many chapters, each chapter signed by the contributing author. The references quoted are voluminous, which the authors state will lead one as far afield as he may wish to go. This work is highly commended to the physician interested in the diseases of the Eye, Ear, Nose and Throat.

The Writing of Medical Papers. By Maud H. Mellish-Wilson, Editor of the Mayo Clinic Publications. Third Edition, Revised. 12mo of 184 pages. Philadelphia and London. W. B. Saunders Company, 1929. Cloth, $1.50 net.

This little volume is recommended to everyone contemplating the preparation of medical articles. For years Mrs. Mellish has been the Mayo authority on such matters.

Surgical Pathology. By William Boyd, M.D., Professor of Pathology, University of Manitoba, Winnipeg Canada. Second Edition, Revised and Reset. Octavo of 933 pages, with 474 illustrations and 15 colored plates. Philadelphia and London: W. B. Saunders Company, March 1929. Cloth, $11.00 net.

This is a most readable work on the very wide field of surgical pathology. The text, the illustrations and the conclusions are based upon knowledge acquired from the built up surgical experience and findings of years past. It will be found most useful to those engaged in surgical work.

Diseases of the Thyroid Gland, by Dr. Arthur E. Hertzler, M. D., Surgeon to the Halstead Hospital, Kansas City, with a chapter on Hospital Management of Goiter Patients by Dr. Victor E. Chesky, M.D., Assistant Surgeon to the Halstead Hospital. Second Addition, entirely re-written. Cloth, illustrated, 286 pages. Price, $7.50, 1929. C. V. Mosby Co., St Louis.

The former issue of Professor Hertzler's work on the Thyroid Gland was popular and well received. This work will be no exception to its predecessor.

Clinical Electrocardiograms; Their Interpretation & Significance by Frederick A. Willius, M.D. Section on Cardiology, The Mayo Clinic, Rochester, Minnesota, and Associate Professor of Medicine, The Mayo Foundation, University of Minnesota. Quarto of 219 pages with 368 illustrations. Philadelphia and London: W. B. Saunders Co., 1929. Cloth $8.00.

Electrocardiographic work is becomming of great importance to the internist and those who specialize in infections of the heart. This book presents the subject in a graphic manner and the text is devoted to the subject of reading records themselves and interpreting their significance.

Modern Methods of Treatment, by Dr. Logan Clendening, M.D., Associate Professor of Medicine, Lecturer on Therapeutics, Medical Department of the University of Kansas, Attending Physician Kansas City General Hospital, Physician to St. Luke's Hospital, Kansas City, Missouri, with pages on several subjects by various collaborators, second edition, illustrated, cloth, 815 pages, Price $10.00. C V Mosby, St Louis.

Dr. Clendening's work on Modern Methods of Treatment will be well received. It will be found of unusual worth to the general practitioner having the responsibility of direction of treatment including medication, diet, and adjuncts. A great deal of attention is paid to the field of various therapeutic measures and to the subject of dietetics.

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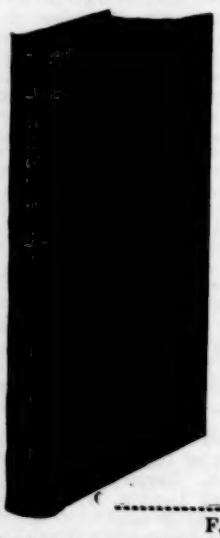

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Muskogee	R. N. Holcombe, Muskogee	A. L. Stocks, Muskogee.
Noble	T. F. Renfrow, Billings	B A. Owen, Perry
Nowata	F. R. Dolson, Nowata	J. R. Collins, Nowata
Okfuskee	A. C. Allen, Weleetka	L. J. Spickard, Okemah
Oklahoma	John F. Kuhn, Oklahoma City	R. L. Murdoch, Oklahoma City
Okmulgee	L. B. Torrance, Okmulgee	M. B. Glismann, Okmulgee
Osage	J. G. Shoun, Fairfax	M. E. Rust, Pawhuska
Ottawa	C. A. McClelland, Miami	J. W. Craig, Miami
Pawnee		
Payne	R E. Waggoner, Stillwater	L. A. Mitchell, Stillwater
Pittsburg	J. A. Munn, McAlester	F. L. Watson, McAlester
Pontotoc	W. H. Lane, Ada	C. F. Needham, Ada
Pottawatomie	Eugene Rice, Shawnee	W. M. Gallaher, Shawnee
Pushmataha	D. W. Connally, Clayton	J. A. Burnett, Dunbar
Rogers	K. D. Jennings, Chelsea	W. A. Howard, Chelsea
Seminole	T. H. Harrison, Wewoka	J. D. McGovern, Wewoka
Stephens	A. M. McMahan, Duncan	J. W. Nieweg, Duncan
Texas	W. J. Risen, Hooker	R. B. Hayes, Guymon
Tillman	J. C. Reynolds, Frederick	J. E. Childers, Tipton
Tulsa	C. T. Hendershot Tulsa	Mary Edna Sipple, Tulsa
Wagoner	S. R. Bates, Wagoner	John D. Leonard, Wagoner
Washington	G. V. Dorsheimer, Dewey	J. V. Athey, Bartlesville
Washita		A. H. Bungardt, Cordell
Woods	C. L. Rogers, Dacoma	O. E. Templin, Alva
Woodward		C. E. Williams, Woodward

NOTE—Corrections and additions to the above list will be cheerfully accepted.